

The Effect of Exercise on the Heart Rate

by

J.A.C. KNOX, M.B., Ch.B.

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(Frontispiece, Script and References).

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¹² They who buy Slaves count their Pulse and Respirations in a given time, and then order them to run ; if now they find the Respiration and Pulse not much altered by that violent Motion, they know that they are of a strong Habit of Body, but the more weak and morbid, the slighter Causes will alter the Pulse, and those who are in a declining way towards Death, have their Pulse twice or thrice as frequent as it ought to be when they exercise the Body. This Experiment is tried by the Officers appointed to purchase Soldiers for our *Dutch Colonies in the East-Indies.*

Frontispiece:

From Dr. BOERHAAVE'S Academical Lectures on the
Theory of Physic, 1757, p. 127.

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effects of varying the intensity, duration and type of exercise were also investigated. The apparatus was found to be very useful for the detection of various cardiovascular abnormalities such as arrhythmias and these are described in the following chapters.

A secondary object was to investigate the possibility of using all or part of this information of the heart rate to judge the condition of the cardiovascular system. In other words, to construct a cardiac index or index of the rather unsatisfactory subject of the heart rate. This was done by constructing a cardiac index on the basis of the heart rate and the body surface area.

PREFACE.

The main object of this research was to find out, in detail, how the heart rate behaves before, during and after mild exertion of very short duration. An apparatus was devised which recorded heart beats during exercise while causing a minimum of interference with the subject. From these records an accurate graph of the heart rate could be constructed and various heart rate indices calculated. The behaviour and variability of such graphs and indices was studied in normal and abnormal subjects. In addition, the effects of varying the intensity, duration and type of exercise were also investigated. The apparatus was found to be very suitable for the detection of various subsidiary factors such as sinus arrhythmia and these are dealt with as they arise.

A secondary object was to investigate the possibility of using all or part of this information on the heart rate to judge the condition of the cardiovascular system; in other words, to construct a cardiac tolerance test. In view of the rather unsatisfactory outcome of the vast amount of research already carried out on this aspect of the subject it was not anticipated that any very positive results would be obtained. Nevertheless it seemed worth while to

try to find out which of the numerous heart rate indices showed the best correlation, even though it might be a poor one, with physical fitness.

The early history of the pulse has been treated in some detail, as it was found that Glasgow University Library was exceptionally rich in the original references to this subject. Photographs of some of these references are included with the permission of the Librarian.

When the more recent literature was reviewed it was found that though an enormous amount of work had been done on the heart rate before and after moderate and strenuous exercise, there was very little on the detailed analysis of its behaviour during and after very light exercises which could be performed without risk by cardiac patients. It was for this reason that the present work, involving the laborious counting of some hundreds of tracings, was undertaken.

Finally, I take this opportunity of acknowledging my debt to those who encouraged the work, and particularly to Professor E.P. Cathcart and Dr. G.H. Bell of the Physiology Department, Glasgow University. The latter collaborated in some of the earlier experiments described here. I should also like to thank Dr. G.A. Allan and Dr. W.R. Snodgrass who allowed me to make use of clinical material from their wards in Glasgow Western Infirmary; and the students and

patients who acted as subjects.

The expense of the apparatus and materials for this research was largely borne by a grant from the Rankin Medical Research Fund of Glasgow University.

"Writers on the pulse may be divided into four classes, namely, those of the Chinese school, those of the school of Galen, those of the school of Solano, and those who attempt to avoid the absurdities of some and the subtleties of others, and to introduce a more natural and simple doctrine". -

Formey, (1823).

CHAPTER 1.

HISTORICAL.

From the point of view of the present study there are three ways in which the history of the pulse may be approached. First, there is the history of the pulse as such, its criteria of normality and its alterations in disease; second, there is the history of the methods of counting and recording the pulse; and third, there is the history of the response of the pulse to exercise. In this brief historical survey it is proposed to take the first two aspects together and to deal with the third separately.

The earliest known reference to the pulse is dealt with in an article by Hamburger (1939). The reference occurs in the Edwin Smith Surgical Papyrus which seems to have been written about 3000 B.C., possibly by the earliest known physician, Imhotep, who flourished in the pyramid age. It appears probable that this physician actually counted the strokes of the pulse, timing them by means of some form of water-clock, and that he realised the connexion between the pulse and the action of the heart. A thousand years later the pulse again appeared in literature, this time in China. The Mo Ching or Pulse Classic (circa. 2000 B.C.) described by Hunter (1938), treated of the complicated ritual to be observed when

feeling the pulse. The great difficulty in counting the pulse which persisted until the 18th century A.D. was due to the lack of an apparatus which could accurately measure small intervals of time. It was not until the second and the minute became practical units of time that the accurate reckoning of pulse rates became possible. The Chinese physicians however got over this difficulty in a most ingenious manner, the physician's rate of breathing being used to count the pulse beats. The physicians appear to have been trained to breathe at a more or less standard rate, and four beats of the pulse to each respiratory cycle was taken as normal.

The next advance took place in the 3rd century B.C. when Herophilus built a water-clock specially for taking pulse rates (Schöne, 1907, quoted by Boas & Goldschmidt, 1932). As Herophilus lived in Egypt he may have been influenced by traditions handed down from the time of Imhotep. The water-clock however was too crude an instrument for accurate pulse counting, and Nicholas de Cusa who attempted to use one as late as the 15th century A.D. found the method difficult and unreliable, according to Wolf (1935). Galen, about A.D. 170 described irregularities in the pulse: "One may be irregular in size, another in rate, another in violence, feebleness and frequency and so on". In his *De Pulsuum Differentiis* he described twenty-seven varieties of pulse with variations

(Finlayson, 1895).

The first man to count the pulse in terms of the minute and the second was the astronomer Kepler. In 1618 he wrote of the pulse: "In a healthy man, robust and of full age, and in one of melancholic complexion, or in a feeble man, generally there is one pulsation of the artery for each second, with no discrimination between systole and diastole; thus there should be in one minute sixty pulsations, but this slowness is rare, commonly seventy may be counted and in the full-blooded and in women eighty, four to each three seconds. Briefly in one hour four thousand more or less". - Kepler (1618). Weir Mitchell (1892) supposed that "the clock with which Kepler counted the pulse must have been such a 'balance' clock as his master Tycho Brahe used". Such clocks, which were capable of beating astronomical seconds, were available only to a very few people; and another invention ascribed to Galileo about 1620 was later developed by Sanctorius for the purpose of pulse counting. This instrument was called the pulsilogon or pulsilogium. The pulsilogium consisted of a leaden bob attached to a long thread and the length of the thread was gradually adjusted until the rate of oscillation of the bob coincided with the pulse rate of the patient. The length of the thread could then be read off in arbitrary figures on a scale attached to the instrument. Comparison of a patient's pulse rate at different times

and of the pulse rates of different patients was thus possible, but the readings were purely arbitrary and there was no reference to a standard unit of time; the instrument was thus an individual and not a universal one.

Harvey (1628), in his "De Motu" made only one mention of pulse rate, saying that in half an hour the heart may make from 1000 to 4000 beats. This estimate clearly shows the difficulty which then existed in making pulse counts.

The next big step forward in pulse counting was made by an English physician, Sir John Floyer. He made universal comparison between pulse rates possible by employing standard units of time. In 1690, according to Wolf (1935), Floyer used a seconds pendulum for measuring the rate of the pulse, and also correlated the pulse rate with the respiration rate. In 1707 he published his "Physician's Pulse Watch" which was the first large scale work on the pulse rate. He noted that "the most natural pulse will have from 70 to 75 per minute in perfect health", and observed that exercise and the passions accelerate the pulse, while sleep slows it. It is interesting to note that he realised the importance of a basal pulse rate:- "by observing the morning pulses before eating, exercise or other external accidents disturb it". Floyer had several pulse watches specially made for him, some of which ran for 60

and others for 30 seconds (Floyer, 1707). But Floyer was ahead of his time and his contemporaries were occupied with far less profitable speculations concerning the pulse.

Thus Nihell (1746) put forward the "doctrine of organic pulses" namely, that the affections of each organ produced a different kind of pulse by which they might be recognised. This was not a new idea however, for it occurred in the Mo Ching (2000 B.C.), and it continued to influence many physicians even into the 19th century.

The normal pulse rate was given by Stephen Hales (1740) as 75 per minute, but in 1749 Van Swieten considered it to be usually 3 pulses in 2 seconds, i.e. 90 per minute. In 1768, Heberden read a paper stressing the value of determinations of pulse frequency, (see Fig. 1, Heberden, 1786), and in the following year Stedman observed that: "we can be sure of the number of pulsations; whereas the various degrees of dilatation of an artery cannot be ascertained with such precision as to establish accurate divisions". He also divided the pulse into classes according to the amplitude and the frequency, and devised an ingenious diagram to represent the various classes (Fig. 2, Stedman, 1769).

In 1796 Falconer summed up and correlated most of the available information on the pulse rate. He agreed with Heberden in saying that "frequency is the only circumstance

respecting the pulse which conveys the same idea to others as to ourselves....." and stressed the need for a general standard for the pulse rate in health (Falconer, 1796).

By the early 19th century, pulse counting had become a more or less routine procedure with physicians, and in 1816 Parry published an account of the nature and cause of the arterial pulse. It is surprising to note that even at this late date he found it necessary to give a detailed account of the views for and against the pulse being due to the systole and diastole of the heart, and incidentally mentioned a case in which the pulse in one radial artery was 90 per minute while in the other it was 180 per minute (Parry, 1816). A few years later Formey pointed out the difference between a quick pulse and a frequent pulse and also noted that according to Solano an intermitting pulse portended a critical diarrhoea. (Formey, 1823).

In 1827 there appeared the two volumes of Rucco's "Introduction to the Science of the Pulse". These were written in an extremely pretentious style and were evidently regarded as the last word on matters concerning the pulse. Rucco was an adherent of 'pure medicine' and regarded the use of instruments as an "irregular and fallacious practice", calculated to distract the attention from all that really mattered in the pulse. He said: "it is only in certain cases

of fevers.....that the use of a watch may sometimes be tolerated during an examination of the pulse," and he condemned all who used such devices as "mechanicians". Rucco also wrote a curious chapter on "the History of Sphygmica" in which he mentions a certain Agathinus who stoutly maintained that the pulse was only perceptible in disease, but never even seems to have taken the trouble to find out whether it did or did not beat perceptibly in healthy persons. Rucco quoted 75 per minute as the normal adult pulse rate, 70 per minute in old age and 60 per minute in "decrepitude" (Rucco, 1827).

In spite of such outbursts against instrumentalism, various physicians and especially Graves and the Dublin school, had firmly established the practice of timing the pulse by the middle of the 19th century (Garrison, 1929). In 1850, as if to mark this stage, the existing knowledge of the pulse was recorded and integrated by Volkmann in his monumental 'Haemodynamik' (Volkmann, 1850).

In 1868 came the first hints of the new methods which were about to be applied to the study of the pulse and the heart. In that year Marey (1868) described the construction and use of his sphygmograph, which gave for the first time an objective measure of the rate and form of the pulse. In that year also the Dutch physiologist Donders first made use of the electric current to aid him in the

timing of the heart-sounds. His method was very simple as there was at that time no direct means of recording the heart beats electrically. By means of a stethoscope he listened to the first and second heart sounds and tapped out their rhythm on a key connected to an electromagnetic marker. The marker recorded their rate and rhythm on a smoked drum along with a time signal (Donders, 1868). Nineteen years later Waller, using a capillary electrometer recording simultaneously with an ordinary cardiographic lever, showed that each heart beat was accompanied by a change of electrical potential (Waller, 1887). This discovery led in 1903 to Einthoven's invention of the string galvanometer and the electrocardiograph which together with the polygraph (Mackenzie, 1893), has played so large a part in the accurate understanding of the mechanism of the heart (Einthoven, 1903). In 1922 the electrocardiograph was used to record the pulse rate during exercise on a treadmill (Smith, 1922).

Three further adaptations of electrical methods must be mentioned, as they were specifically designed for the recording of heart rate. The first of these was really a modern version of the old 'pulsilogon', and was called the 'pulse resonator'. The pulse wave was employed to make and break a circuit which activated an electromagnet. Around this magnet were arranged a series of pendulums of

different periods which were set in motion by the impulses from the electromagnet. Each pendulum thus corresponded to a particular pulse rate and according to which pendulum was oscillating a recording device wrote the curve of pulse rate on moving paper (Kraus, Goldschmidt & Seelig, 1926). A disadvantage was that the subject could not move about but had to remain in a fixed position. Nemet & Boas (1928), by checking tracings made with the pulse resonator against simultaneous electrocardiographic records; showed that the apparatus, though fairly accurate over long periods, would not respond to rapid changes in heart-rate.

Two years later Boas published the details of his 'cardiotachometer'. This instrument was designed to count the total number of heart beats over long periods of time, and was operated by the action-current of the heart itself. Chest electrodes were used and the action-current was amplified by valves until it was powerful enough to operate an electromagnetic relay, which in turn operated a cyclometer, the heart beats being thus automatically counted over any given period. A signal, also operated from the relay, caused each beat to be marked on a moving paper strip. The great advantage of this apparatus was that the subject could move about freely without upsetting the recording mechanism. (Boas, 1928).

The third apparatus for recording pulse rate was Fleisch's 'Pulszeitschreiber', first described in 1930. In this instrument a pelotte strapped to the wrist was used to make and break an electric circuit with each pulse wave. A writing point was moved vertically upwards by an electric motor during each pulse interval, and when each pulse wave arrived, the writing point was declutched from the motor and fell back to zero. Thus the slower the pulse rate, the longer was the vertical line which indicated the duration of each pulse interval. As the wrist had to be strapped into a fixed frame, however, the subject's movements were very limited. Full descriptions of the apparatus were given by Fleisch (1930) and in Abderhalden (1935). A somewhat similar apparatus for recording any rhythmic process had previously been described by Loomis & Harvey (1929). In 1935 Whitehorn, Kaufman & Thomas combined the Fleisch Pulszeitschreiber and the Boas cardi tachometer into an instrument which they called the cardiochronograph.

The Response of the Pulse to Exercise.

The fact that exercise increases the pulse rate must have been known from a very early period, and a crude but useful cardiac tolerance test appears to have been used by enterprising slave traders about the beginning of the eighteenth century or even earlier. Boerhaave (1757) in his "Theory of Physic" gave an interesting account of this practice (see facsimile reproduction in frontispiece). In the year 1707 Sir John Floyer, using his "Pulse Watch", made quantitative observations on the accelerating effect of muscular exercise on the pulse rate; and as was noted above, emphasised the importance of starting with a basal level.

Twenty-five years later Bryan Robinson published further quantitative results, stating that the pulses in a minute of a man lying, sitting, standing, walking at the rate of two miles an hour, and running as fast as he could, were 64, 68, 78, 100 and 150 or more, respectively. He also noted that: "When a Body stands up, the Pulse begins to grow quicker the very Instant the Body begins to rise, or the Soul begins to exercise the Power which raises it....." (Robinson, 1732). It was recognised however that the pulse alone, apart from exercise tests, was insufficient as an indicator of physical condition and in 1786 Heberden wrote; "from these remarks it appears that the pulse, though in

many cases an useful index of the state of the health, yet it is no certain one in all; and that without a due regard to other signs it may mislead us".

Experiments carried out three years later by Seguin & Lavoisier showed an interesting anticipation of modern methods. The pulse was counted during experiments on the gaseous metabolism of a man performing exercise, both in the fasting state and during digestion. Weight-lifting was the exercise used, and the authors came to the conclusion that the augmentation of the number of pulsations of the arteries was directly proportional to the total weight lifted to a given height, provided the subject was not pushed to the limit of his endurance. They also concluded that the amount of vital air consumed was directly proportional to the product of the number of inspirations and the pulse rate: a remarkable piece of research. (Seguin & Lavoisier, 1789).

Falconer (1796) observed that the average difference between the pulse rates in the sitting and standing postures was: "about six beats and one third in a minute". He also tried experiments on the effects of walking and running; "but the result in each of them was so different that I could not reduce them to any standard". Robert Knox, the famous Edinburgh anatomist, also carried out researches on: "The Manner in which the Pulsations of the Heart and

Arteries are affected by Muscular Exertion". He found that the pulse was both quicker and more excitable in the morning than at any other time of the day or night; and attempted to correlate this finding with the observation of Prout (1813) that the greatest quantity of carbonic acid gas, formed during respiration, was generally given off during the morning hours. Nevertheless, his final conclusion was; "that the quantity of carbonic acid given off during respiration is not particularly connected with, or at all events not dependent on, the state of the circulation". (Knox, 1815).

With regard to the mechanics of the circulation, Rucco (1827) gave a remarkable account of the phenomena which are now explained by the 'Bainbridge Reflex' and by 'Starling's Law of the Heart'. A facsimile of the passage in which he visualises the blood rushing into the heart during muscular exercise, causing the ventricles to beat rapidly and forcibly; "in order to disembarass themselves of this accumulation of blood", is given in Fig. 3.

Up to this time walking and running, often over considerable distances, were the usual forms of exercise employed by workers on the pulse rate; but Donders (1868) used the rapid ascent of stairs as a more convenient and controllable form of activity. The merits of this form of exercise were further developed and stressed by Selig (1905,

quoted by Master & Oppenheimer, 1929). With the careful and detailed work of Bowen (1903) and of Selig began the modern study of heart rate during exercise. The results of later workers in this field will be dealt with in the succeeding pages.

The first of the early workers who used objective means for the study of pulse rate. At first there were only a few workers who used the pulse rate as a measure of heart rate.

The effect of exercise on the pulse rate was investigated as soon as suitable instruments became available. A summary of some early work on this subject is given below.

SUMMARY.

A brief survey of the early history of the pulse is given. For obvious reasons the discovery of the significance of the pulse rate is closely linked with the development of time measuring devices. Even when suitable watches were available there was opposition to the practice of pulse counting. Much later came the objective methods of recording the pulse rate. At first these were mechanical but subsequently electrical methods were developed.

The effect of exercise on the pulse rate was investigated as soon as suitable instruments became available and a summary of some early work on this subject is included.

"Among the various signs of diseases, none are more frequently, nor more justly, attended to than those arising from the pulse; and though such may be, for the most part, insufficient by themselves to ascertain the nature of any distemper, yet we can seldom, with safety, proceed to the cure, without taking some indication from the state of the pulse".

Stedman (1769) p.2.

CHAPTER 2.

CONTACT ELECTROLYTES IN ELECTROCARDIOGRAPHY.

As will be seen from the next chapter, the method of recording the heart beats during exercise employed throughout this study is a modification of ordinary electrocardiographic technique. It was soon found, however, that for steady and accurate recording it was essential to have really good contact between the subject's skin and the electrodes which picked up the action current of the heart.

A very common clinical method of making this contact is to wrap a few layers of gauze soaked in saline round the limb and to bind a metal electrode over the gauze. A disadvantage of this method is that the limb to limb resistance is rather high and increases considerably as the gauze dries. Also, the bedclothes must be protected from becoming wet when an electrocardiogram has to be taken from a bedridden patient. If the limb to limb resistance is high the string of the Einthoven galvanometer must be slackened to increase the sensitivity, and so the record may be distorted by the alteration of the damping and the reduction of the natural frequency of the string (Pardee, 1917). In the case of valve electrocardiographs, with their high input resistance - usually about 100 times that of the string - high body resistance is not so disadvantageous; but if it can be kept low there is less likelihood of electrical

interference, and in addition the stability of the amplifier is increased.

In the early experiments with the present method of recording, such saline pads were tried, but as some of the tests lasted more than an hour, considerable trouble was experienced owing to the drying of the electrolyte. Boas (1928) recommended ordinary soft green soap as a contact electrolyte and this was found to fit the present requirements admirably.

Although skin resistance has often been investigated in connection with chronaxie and the psychogalvanic reflex, no report could be found of a systematic investigation into the properties of contact electrolytes, apart from the work of Hartridge (1931) which dealt with the effect of grease-removing substances. It was thought, therefore, that it would be of interest to compare soft green soap with certain other electrolytes.

The subjects were myself, Dr. G.H. Bell of the Physiology Department and Dr. A.J. Small of the Electrical Engineering Department, who collaborated in this investigation (Bell, Knox & Small, 1939).

Method.

The electrolytes investigated were:

1. One per cent. sodium chloride in tap-water.
2. Soft green soap (Sapo Mollis Viridis, B.P.) which consists chiefly of an almost neutral mixture of potassium oleate with a little glycerine.
3. Cambridge electrode jelly, which is sold by the Cambridge Instrument Co.; the composition of this substance is not issued.
4. A paste described by Jenks & Graybiel (1935) which will be called, for convenience, Boston paste; it consists mainly of sodium chloride, glycerine, water, and powdered pumice.

In the case of the first electrolyte, two layers of gauze bandage soaked in saline were wrapped round the forearms. Plymet electrodes each 14 cm. by 5 cm. were laid on the volar surfaces and held in place by rubber bands. Plymet is a soft sheet metal (supplied by Schall and Son, New Cavendish Street, London, W.1.), consisting mainly of lead, which is readily moulded to the shape of the arm.

In the case of Cambridge jelly a piece of the jelly about the size of the finger-nail was rubbed into the forearms for about ten seconds, as recommended by Russell (1935). The plymet electrodes were then smeared with the jelly, put directly on the skin, and held in place with

bandages and rubber bands. The soft green soap and Boston paste were applied in a similar manner.

As is well known, there are some quick changes (rise and fall of R wave) and some slow changes (T wave) in the electrocardiogram. Hence an adequate description of the behaviour of an electrolyte should include both the D.C. resistance and the A.C. impedance.

The measurements were made by means of a bridge in which the subject formed one arm, and a variable resistance with a variable capacitance in parallel formed the second arm; the ratio arms were formed by two equal resistances of 1000 ohms. Alternating current was supplied to the bridge by a valve oscillator which gave about 8 volts at 300 cycles per second (c.p.s.). A sensitive vibration galvanometer tuned to 300 c.p.s. was used to find the balance condition. The values of resistance and capacitance in the second arm, combined vectorially, gave the A.C. impedance, Z.

Thus
$$Z = \frac{r}{\sqrt{1 + \omega^2 C^2 r^2}}, \text{ where } \omega = 2\pi f$$

When the A.C. measurement had been made, an 8-volt battery was substituted for the oscillator and a moving coil galvanometer for the vibration galvanometer and the bridge brought once more to balance by varying the resistance in the second arm. This gave the direct current resistance, R.

Results.

The wide scatter of the results, which are collected together in Fig. 4, is not altogether surprising in an experiment of this nature. As it was not, of course, possible to get simultaneous values for the different electrolytes, they were applied in turn, varying the order with each subject.

The values of Z were distributed over a considerable range. One per cent. sodium chloride gave, on the whole, the lowest values (average 652 ohms), Boston paste gave slightly higher values (average 689 ohms), whilst green soap and Cambridge jelly both gave still higher values (average 768 and 754 ohms respectively).

R was found to be highest with 1 per cent. sodium chloride, the average value being 3080 ohms. Cambridge jelly and green soap gave the somewhat lower average values of 2010 and 2040 ohms respectively. Boston paste gave the lowest average resistance of 1100 ohms.

In order to allow of further discrimination between the solid electrolytes, variation of R and Z with time was investigated. The electrodes were left in position for varying times up to one and three quarter hours and Z and R, in that order, were measured at quarter-hour intervals. The leads were removed from the bridge between readings. In all cases Z and R rose steadily, but not very greatly, for about three quarters of an hour after application of the

electrodes, when the values became nearly constant (see Fig. 5).

Discussion.

From the wide scatter of the results shown in Fig. 4 it will be seen that it is not at all easy to choose the best electrolyte on the basis of Z and R values only. The best is probably Boston paste. Green soap and Cambridge jelly are only slightly inferior; sodium chloride, on account of its very high R, is the poorest.

Drying up of the solid electrolytes does not occur to any great extent because none of them show any great increase in resistance up to one hour or more after application. There was no difference between electrocardiograms taken by means of a string galvanometer using first green soap and then Cambridge jelly. The choice between the three solid electrolytes will, therefore, have to be made on other grounds.

As green soap is a standard British Pharmacopoeial substance and costs only about one penny per oz., it is to be preferred to the complicated Boston paste or the expensive Cambridge jelly.

The Effect of Abrasives.

Boston paste is made up with a considerable proportion of powdered pumice, which at first was regarded merely as an inert base required to form the paste. But

examination of Cambridge jelly points to a different conclusion. It appears to consist chiefly of sodium chloride in a base which is probably not lanoline or petroleum jelly, but which may be a gum. The jelly feels gritty to the touch, and on dissolving away the base in hydrochloric acid an insoluble residue remains, which under the microscope proves to consist of extremely jagged particles of crushed quartz.

It is now clear why the jelly should be rubbed into the skin; the sharp particles remove the surface cells and so contact is made with a deeper layer. Richter (1926a and 1926b) showed that a minute puncture of the skin below electrodes one inch square reduced R from 540,000 ohms to 15,000 ohms. Lewis & Zotterman (1927) came to the conclusion that the high resistance displayed by skin to galvanic currents resided in the superficial and horny layer. Accordingly it was decided to investigate the effects of abrasion quantitatively.

Method and Results.

To one sample of green soap pumice was added in the proportion occurring in Boston paste and to another sample crushed quartz recovered from Cambridge jelly.

In these experiments the average value with green soap alone for Z was 885 ohms and for R was 2450 ohms. After rubbing in green soap with quartz the average values

were Z 665 ohms and R 890 ohms, a decrease of 25 per cent. in Z and 64 per cent. in R. Rubbing in green soap with pumice left Z unchanged but reduced R by 57 per cent. Green soap covers the quartz and pumice particles and interferes with their abrasive qualities. It was probable, therefore, that it would be more effective to abrade the dry skin and then apply the electrolyte and electrode. The surface of the skin was rubbed gently two or three times with fine glass paper (No. 1). This raised the surface layer of cells so that the skin lost its sheen and became white in colour. This procedure resulted in a reduction of Z by 60 per cent. and of R by 80 per cent. on the average, the mean values after such abrasion being; Z, 400 ohms, and R, 500 ohms, approximately. These were the lowest values observed during the whole course of this investigation.

In addition to the alteration of Z and R brought about by abrasion, it was noted in measuring Z that the resistive component required for balance fell to about one-half, or less, of the value before abrasion. The capacitance in parallel, however, fell to about one-tenth. For example, in one experiment before abrasion the bridge was balanced by 1790 ohms in parallel with 0.386 mfd., i.e. $Z = 1090$ ohms; after abrasion, balance was given by 368 ohms in parallel with 0.035 mfd., i.e. $Z = 368$ ohms.

Discussion.

From the above results there is no doubt that if the lowest possible body resistance is required, gentle abrasion of the dry skin with glass paper, followed by application of green soap and then of the electrodes is the most convenient method. Provided the rubbing is gentle and only two or three strokes are used, the skin will return to its normal appearance in a few hours. There seems to be nothing objectionable in making this a routine procedure, provided that no irritating antiseptics, such as iodine, are applied afterwards. Only one minor difficulty appeared; it is rather hard to use glass paper on hairy parts, because the hairs roll round under the paper and no abrasion of the skin is made. This could be overcome by removing the hairs before abrasion. It was found that more vigorous glass papering, or scratching, which left a mark taking over a week to disappear, did not give lower values than the more gentle method.

By the use of the procedure recommended above the external resistance (i.e. of the patient) becomes a much smaller fraction of the resistance of the galvanometer, which should then be expected to give a better record of the potentials occurring in the patient's body. Also, when the patient's resistance is low the electrocardiograms obtained should be more standardised, as even relatively large

alterations of this resistance will then have little effect on the total resistance of the circuit. It was found that in the case of the A.C. bridge the capacitance value (without abrasion) varied from 0.15 to 0.5 mfd., using the same electrodes. It is difficult to predict what would be the effect of this capacitance on the electrocardiogram. The effect of capacitances across the string on damping has been studied by Gildemeister (1922), Dock (1928) and Schwarzschild & Kissin (1934). Here again, however, more standard results would be expected when the capacitance is low, as is the case when the skin is abraded, because the variations in the capacitance then become of small importance. It was also noted that when the skin was abraded the values of R and Z tended to be very steady, whereas without abrasion there was a tendency to drift.

It is interesting to speculate how far the differences which are occasionally found between records taken by string and valve electrocardiographs are dependent on high patient resistance and impedance. There seems to be a tendency to regard records taken by a string galvanometer as standard, in spite of its relatively low resistance. This tendency seems to be based more on tradition than on pure reason. It would be interesting to compare string and valve electrocardiograms in a series of cases taken with the technique described here. It is probable that with the low

patient resistances so obtained a number of the discrepancies would disappear. This question has already had some attention directed to it by Pardee (1929).

Variations in the Quality of the Green Soap.

During the course of these experiments, three separate samples of Sapo Mollis B.P. were purchased. The first sample was soft, bright green in colour, and spread very easily and evenly over the skin surface. The second sample was harder, dark green in colour, very tenacious, and became caked when rubbed on the skin, refusing to spread evenly. The following figures show that this second sample was inferior to the first for our purposes. The values for R were usually over 10,000 ohms, and the average value for Z (11 observations) was 915 ohms, compared with the averages R 2040 ohms and Z 768 ohms for the first sample.

The third sample of Sapo Mollis was comparable in every way with the first, and in obtaining it the following criteria were observed. The green soap must be fresh and should be bright green in colour, and it should spread as easily on the skin as a good ointment base. It should be kept in an air-tight tin or waxed carton.

The difference in effectiveness was not due to a change in the resistance of the soap. The specific resistance of the second sample (tough soap) was 18.4 ohms per cm. cube; the value for the third sample (good soap) was 23.9 ohms per

cm. cube. It is to be presumed that the lowering of skin resistance with the good soap is due to the fact that it spreads so easily and evenly over the skin.

1938).

The arm to arm D.C. resistances (R) and impedances (Z) vary over a wide range for all electrolytes. From this point of view the best is the only Boston paste, but Cambridge jelly and most other soaps are very inferior.

It is cheap, clear, simple, and a standard soft green soap spread easily over the skin. It should be the best, ought to be the standard.

The Boston paste and the Cambridge jelly are the best for operation of an electrolytic cell. The storage values of electrolytes are to a considerable extent.

The electrolytic cell were changed to the electrolytic cell of the electrolytic cell.

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SUMMARY.

Reasons are given for the necessity of having a low limb to limb resistance when obtaining electrocardiograms either by the string galvanometer or by a valve electrocardiograph.

A comparison has been made between four electrolytes, viz. 1 per cent. sodium chloride, soft green soap, Cambridge electrode jelly, and Boston paste (Jenks & Graybiel, 1935).

The arm to arm D.C. resistances (R) and impedances at 300 cycles per second (Z) vary over a wide range for all these electrolytes. From this point of view the best is probably Boston paste, but Cambridge jelly and soft green soap are very little inferior.

Because it is cheap, clean, simple, and a standard pharmacopoeial product soft green soap appears to be the most satisfactory. It should be fresh, bright green in colour, and should spread easily over the skin.

Both the Boston paste and the Cambridge jelly contain abrasives. Incorporation of an abrasive in the green soap reduced the average values of resistance and impedance to a moderate extent.

The lowest values of limb to limb resistance (R, 500 ohms; Z, 400 ohms) were obtained when the abrasion preceded the application of the electrolyte as follows:

the dry skin was stroked gently two or three times with fine glass-paper, green soap was then applied and the electrodes were bound on.

"The having recourse, as several physicians are accustomed to do, to an artificial standard, such as a watch, pulsilogium, or pendulum, as a substitute for the natural one, afforded by the exact knowledge of the physiological state of the pulse, is an irregular and fallacious practice, for the evident reason, that by means of the former it can only be ascertained how often the artery pulsates in one or more minutes, and whether the pulse be frequent or rare, quick or slow; but in no way can it be ascertained, if the pulse be strong or weak, great or small, hard or soft, equal or unequal, critical, organic, or symptomatic".

Rucco, (1827) p.35.

CHAPTER 3.

METHOD OF RECORDING THE HEART BEATS.

The essential points of the method have already been published (Bell & Knox, 1938) but no detailed description of the apparatus at present used has been given.

Apparatus:- The apparatus consists essentially of three parts; the leads from the subject, the amplifier, and the writing mechanism.

The Leads from the Subject:

In order to pick up the action current from the subject's heart, two chest electrodes are employed. The electrodes are saucer-shaped copper discs 2.5 cms. in diameter. The concavity is filled with soft green soap which acts as the electrolyte, and the electrodes are kept in contact with the chest by means of adjustable light elastic straps. One electrode is applied over the apex-beat and the other over the second right costal cartilage. With the electrodes in these positions a satisfactory voltage and an even base line are obtained. The respirations are recorded by means of an ordinary stethograph tied lightly round the subject's chest and connected by thin rubber tubing to the recording tambour. In order to relieve the subject of the drag of the flex and rubber tube, these are slung from a 'trapeze' which swings over the subject's head.

A general view of the apparatus showing the leads is given in Fig. 6.

The Amplifier.

A three-stage resistance-capacity-coupled circuit is used (Fig. 7) with balanced input in order to reduce outside interference to a minimum. The time-constant of the intervalve coupling condensers is reduced very considerably below the normal value as this minimises the effects of skeletal muscle activity. The slow T and P waves of the electrocardiogram are therefore not reproduced on the tracing, but this is no disadvantage when records of pulse rate only are required. Alternative switching to larger coupling condensers is provided so that ordinary electrocardiograms may also be taken by means of a Matthews Oscillograph.

The Writing Mechanism:

Fig. 8 shows the writing mechanism and drum. It will be seen that four writing points are applied to the drum and that they all lie in the same vertical line. This of course compensates for any slight unevenness in the running of the drum mechanism.

The uppermost lever is pneumatically operated and is used as a signal to indicate the beginning and end of exercise, etc.

The second writing lever is connected to an electromagnetic time-marker controlled by a Brodie clock which beats seconds or half-seconds.

The third lever consists of a stiff glass pointer with a ball point connected to the arm of a Weston moving-coil relay modified as described by Winton (1936) and Bell, Bell, Knox & Smellie (1937). The relay is connected in the output circuit of the amplifier and the pointer is thus made to record the subject's heart-beats on the smoked drum. It is to be noted that though this Weston instrument was manufactured for use as a relay, it is used here simply as a moving-coil galvanometer, thus preserving electrical continuity with the cardiac action currents.

The fourth writing point records the subject's respiratory movements by means of an ordinary tambour connected to the stethograph on the subject's chest. The lever moves downwards on inspiration and upwards on expiration. A typical tracing is shown in Fig. 9.

SUMMARY .

The main advantages of the method are:-

- (1) It allows the heart beats to be recorded without interruption even during violent exercise of any group of muscles.
- (2) It has proved reliable over a considerable period of time.
- (3) It is cheap, as no expensive photographic materials are required.
- (4) The apparatus is so constructed that the recording galvanometer is in direct electrical continuity with the action-current of the heart. Thus the question of 'losing touch' with the heart-beats does not arise; and if, for example, a premature beat of low voltage occurs it is simply recorded as a smaller stroke on the drum. The somewhat similar types of apparatus in which a relay mechanism is used to operate the recording device are open to the objection that a beat of low voltage may fail to operate the relay and thus may not be recorded at all. For example, the cardi tachometer devised by Boas (1928) has this disadvantage.

"Praxagoras, moreover, fell into the error of making so many divisions and subdivisions of the pulse, that, according to Galen, they formed a collection of obscure and unintelligible enigmas, rather than a body of learned observations....."

Rucco, (1827) p. 53.

CHAPTER 4.

THE METHOD OF ANALYSING THE TRACINGS.

The apparatus was used to record the heart beats during a standard exercise which will be described in the next chapter, the changes in heart rate being determined by analysing the tracings in the following manner.

In order that the heart beats may be accurately counted, fine vertical lines are drawn across the tracings at intervals of five seconds. The first line is drawn exactly ten seconds before exercise begins and the lines are continued until about twenty-five seconds after exercise ends. (Black pencil lines A, B, C, D, etc. in Fig. 10). The heart rate is then counted (to the nearest tenth of a beat) in each of these five-second intervals, and the highest rate obtained is converted to beats per minute and is taken as the MAXIMUM RATE. The time from the beginning of exercise to the middle of the five-second period containing the maximum rate is taken as a measure of the TIME TO REACH THE MAXIMUM RATE. For example, if the maximum rate is found in the third five-second period from the beginning of the exercise, the time to reach the maximum rate is 12.5 seconds.

The heart rate in the 10 seconds immediately preceding the beginning of the exercise, converted to beats

per minute, is taken as the INITIAL RATE. (Lines A to C, Fig. 10).

The ACTUAL INCREASE (in beats per minute) of the maximum rate over the initial rate is given, and this is also expressed as a percentage of the initial rate (PERCENTAGE INCREASE ON INITIAL RATE). The ACCELERATION OF THE HEART RATE in beats per minute per second is calculated by subtracting the rate in the five-second period immediately preceding exercise (lines B to C in Fig. 10) from the maximum rate and dividing the result by the time taken to reach the maximum rate.

Another index which may prove of interest is the NUMBER OF EXTRA HEART BEATS induced in a given time by the exercise. This is calculated by subtracting half the initial rate (in beats per minute) from the number of beats in the 30 seconds following the beginning of exercise (lines C to K in Fig. 10) which includes the whole of the exercise period.

In order to have an index which does not require the use of apparatus, the heart rate is counted over the 30 second period beginning 5 seconds after exercise ends (red lines X to Y in Fig. 10). This is expressed in beats per minute and is called the POST EXERCISE RATE.

An index which has been much employed in tolerance tests is the time which the pulse rate takes to return to

normal after exercise. This is usually obtained by counting the rate at minute or half-minute intervals until the pre-exercise rate is reached. The present method of recording however, offers an opportunity of determining the time of return to normal within very narrow limits, and it was considered that it might be interesting to see whether this index was of any value when determined by what might be called a "micro" method. The procedure adopted is as follows:- The length of tracing occupied by the last ten beats before exercise begins is marked off on the edge of a sheet of paper (distance M-N in Fig. 10). The paper is then slid along the tracing until the next group of ten beats which will just fit this space is encountered (group O-P in Fig. 10. Distance M-N is, of course, exactly the same as distance O-P). The time from the end of exercise to the beginning of this second group of ten beats is taken as the TIME TO RETURN TO NORMAL, (time from E to R in Fig. 10). In addition to the above indices the rate is counted for 30 seconds in the middle of the 5 minute rest period before exercise. This count, which cannot be shown in Fig.10 owing to lack of space, is called the EARLY RESTING RATE and is expressed in beats per minute.

As some of the above procedures appear rather complex at first sight, it may be useful at this stage to

give an example.

Example of the Analysis of a Typical Tracing.

The tracing in Fig. 10 is here analysed and the letters refer to the lines in that figure.

Early Resting Rate (not shown in figure) = 33 beats in 30 sec.
i.e. 66 beats/min.

The heart beats in the successive 5 second periods, starting 10 seconds before the beginning of exercise, are as follows:-

5.3, 6.0, 8.3, 9.1, 9.3, 9.6, 9.1, 8.4, 7.5, 7.2.

The Duration of Exercise (lines C to E) is 19.5 seconds.

The Initial Rate (lines A to C) is $11.3 \times 6 = 67.8$ beats per minute.

The Maximum Rate is $9.6 \times 12 = 115.2$ beats per minute, and it occurs in the fourth 5 second period after the beginning of exercise; therefore the time to reach the maximum rate is taken as 17.5 seconds.

The Actual Increase in rate is Max. Rate minus Initial Rate, i.e. $115.2 - 67.8 = 47.4$ beats per minute.

The Percentage Increase on the initial rate is

$$\frac{\text{Actual Increase}}{\text{Initial Rate}} \times 100, \text{ i.e. } \frac{47.4}{67.8} \times 100 = 70\%$$

The Acceleration of the heart rate is

Max. Rate minus rate in 5 sec. period immediately before ex.,
Time to reach max. rate

$$\text{i.e. } \frac{115.2 - 72}{17.5} = 2.47 \text{ beats per minute per second.}$$

The number of beats in the 30 seconds after exercise began
(lines C - K) is 54. Thus the number of extra beats induced
the exercise is 54 minus Initial Rate,

$$\text{i.e. } 54 - \frac{67.8}{2} = 20.1 \text{ beats.}$$

The Post-Exercise rate (lines X to Y) is 43 beats in 30
seconds = 86 beats per minute.

The Return to Normal (lines E to R) is 52 seconds.

Accuracy of the Method.

As regards the accuracy with which the vertical lines can be drawn and the heart beats counted, a somewhat similar device was adopted by Smith (1922) and by Cotton & Dill (1935). They found that errors of draughtmanship (erecting vertical lines and judging tenths of a beat interval) were negligible. The same was found to hold in the present case.

SUMMARY .

The method of obtaining the various heart rate indices which will be employed throughout this research is given, with an example. The accuracy of the method is also briefly discussed.

"The pulse is liable to vary from so many different circumstances as must necessarily render such calculations inaccurate, and supposing that the pulse could be examined freed from these embarrassments, it is well known that the natural pulse in different individuals varies considerably, and of course, what may serve as a standard of computation in one instance may prove very erroneous in another. It is nevertheless perhaps possible to adjust such allowances, as may bring these varieties within such limits as may serve to fulfil in a great measure most of the purposes of medicine, however insufficient they may appear, to lay the foundation of any regular system of physiology or pathology".

Falconer, (1796) p.4.

CHAPTER 5.

THE STANDARD EXERCISE.

Though the primary object of the research was to determine in detail the changes in heart rate during and after exercise in normal and abnormal subjects, the possibility that some of the data obtained might serve as indices of cardiovascular fitness was not overlooked. Before coming to a final decision on the type of exercise to be employed, the literature on cardiac tolerance tests in general was reviewed.

The opinions of cardiologists as to the value of exercise tolerance tests in estimating the functional efficiency of the heart have varied considerably. At first too much was hoped for, then came a period of disillusionment, and recently the Horder Committee (1940) has reinstated a revised form of exercise tolerance test as a useful adjunct to a full cardiac examination.

The choice of an exercise tolerance test which would fulfil the objects of this research proved far from easy. The literature showed that almost every conceivable form and grade of muscular activity had its sponsors and its critics. There were exercises of speed, of endurance and of strength; there were resistance exercises and postural exercises, and the results were variously interpreted in terms of pulse-rates, blood-pressures,

heart sizes, vital capacities and so on almost indefinitely.

A further complication was that some tests claimed to be "cardiovascular function tests" pure and simple; while others purported to measure "physical fitness" or "physical efficiency", and these latter, according to White (1920 and 1937), are measures of training and of the nervous state rather than of cardiac condition. Bainbridge (1919) however, pointed out that the functional capacity of the heart determined a man's capability for exertion, and Burns (1939) stated that vaso-cardiac efficiency was the real limiting factor in physical fitness. Thus any sharp distinction between cardiac functional tests and physical fitness tests is obviously artificial. The whole question of the assessment of 'fitness' has been reviewed by Magnus-Alsleben (1924), Hambly, Pembrey & Warner (1925), McSwiney (1939) and Cathcart (1943).

Evans (1912) using an isolated heart-lung preparation found that the rate of gaseous metabolism varied almost exactly as the heart-rate and the observations of Benedict & Cathcart (1913), Benedict (1915) and Benedict & Murschhauser (1915) established this close relationship between pulse frequency and metabolism for man, though their data referred mainly to the alterations in metabolism of single subjects. This close relationship, which has been confirmed by the work of Henderson (1925), Paterson (1928),

and Griffith et al. (1929), does much to encourage the hope that the pulse rate may yet be found to be a reliable mirror of cardiovascular reactions. Addis (1922) has pointed out, however, that the relationship is probably a more general one between the metabolism of the body as a whole and the activity of the circulatory system, of which the pulse rate is only a partial expression.

The following is a brief summary of the various types of heart function test which have been tried. Kahn (1919) introduced hopping one hundred times on one foot with observation of the effects on the pulse rate and blood-pressure, and various modifications of this test are still used clinically (Lewis, 1945) as it has the advantage of requiring the minimum of apparatus. Barringer & Teschner (1915) introduced a test in which the subject raised a pair of dumbbells with each arm alternately, and employed the 'delayed rise' in systolic blood pressure after such exercise as a measure of functional capacity. This type of test was further expanded by Barringer (1916 and 1917), and was found valuable in the assessment of convalescents by Mann (1918). Later, Barringer (1922) suggested that the so-called 'delayed rise' in blood pressure signified that the heart was being overtaxed, irrespective of the exercise employed. According to Rapport (1917), Propst (1924), and White (1937) however, this delayed rise of pressure is of little or no

significance. Dumbell exercises were also used by Mabon (1919), Wilson (1920 and 1921) and by Brittingham & White (1922). Barach's "energy index" $(SBP + DBP) \times P.R.$ was also proposed as the basis of a fitness test (Barach, 1916). Another type of test attempted to assess cardiac efficiency by measuring the alterations in heart rate and blood pressure when the subject changed from the horizontal to the vertical posture. Hill, Barnard & Soltan (1897) attempted to measure fatigue by this means. According to Vierordt (1906) the average postural increase in heart rate is from 12 - 14 beats/minute, and Crampton (1915) made use of this increase and of the normal rise of S.B.P. of about 10 mm. of mercury in his 'blood ptosis test'. Schneider & Truesdell (1922a) found a smaller increase in pulse rate when changing from lying to standing in a fit group than in an unfit group.

A very popular type of test is the 'step test'. Many varieties of this are in use but in all of them the exercise consists in the climbing of stairs or steps a specified number of times. Donders (1868) appears to have been one of the first to adopt this particular form of exercise. Among those who have made use of step tests may be mentioned Christ (1894), Staehelin (1897), Lewis (1917), Flack & Bowdler (1920), Wilson (1921), Schneider & Truesdell (1922a), Cripps (1924), Woolham (1924), Campbell (1925),

Woolham & Honeyburn (1927) and Schott (1939). Exercises on steps were also used by Hunt & Pembrey (1921) in their 'pulse-ratio' test for physical efficiency. An extremely convenient form of step test which will require further mention was devised by Master & Oppenheimer (1929) and was further expanded by Master (1934-35). The apparatus consisted of two steps each nine inches high, so that at each climb the subject raised himself $1\frac{1}{2}$ feet above the ground. The blood pressure and pulse rate were taken before and two minutes after the exercise and the latter readings should normally be within 10 points of the former. The number of ascents to be performed was obtained from tables and depended on the subject's age and weight. Reisinger (1938) however, did not find the Master test entirely satisfactory and concluded that a diagnosis of heart disease could not be made on the results of this test alone, and Kuskin & Brockman (1940) found no relationship between performance in the two-step test and the extent of cardiac damage. In 1942, Master, Friedman & Dack examined the electrocardiographic changes in normals and anginal patients after the Master test and found it to be a good measure of cardiac function and this was confirmed by Goldbloom & Dumanis (1945).

Tests of cardiac function and physical fitness which do not depend on the counting of the heart-rate have

also been devised, and as examples of these may be cited the 'vital capacity' tests of Dreyer & Hanson (1920), Brittingham & White (1922) and Hewlett (1924). In these tests tables of normal vital capacities depending on the height and chest measurement of the subject are given, and if the vital capacity falls far short of normal it is taken as an indication of diminished functional efficiency. A method of measuring fitness has also been described which depends on the divergence of the curves obtained when exhaled CO₂ percentage is plotted against work done when breathing air and when breathing oxygen (Briggs, 1921), while a metabolic test based on excess oxygen consumption during exercise was devised by Katz et al (1934).

Teleroentgen tests, which measure the alterations in size of the heart as a result of exercise, have also been used. It has been said that the normal heart responds to any exercise within its power by a prompt diminution in size and that this may be used as a test of its functional efficiency (Williamson, 1915). The reactions of heart rate and blood pressure to inhalation of amyl nitrite (Cotton, Slade & Lewis, 1917); and to exposure to low oxygen tensions (Schneider, 1918 and Whitney, 1918) have also been suggested as heart function tests, but the latter type of test was not found to be trustworthy by Stengel, Wolferth & Jonas (1920)

and Schneider himself reported unfavourably upon it in 1921.

The last type of test which must be mentioned may conveniently be termed the "Multiple Test", and the principle underlying it is simple. The subject performs a number of function tests chosen from the range indicated above and marks are allotted for each, The subject is then graded according to the total number of marks obtained. Schneider (1920) was one of the first to use this type of test and his system of 'cardiovascular rating' was based on the response of the pulse rate and blood pressure to postural changes and to a step test. He emphasised that the rating was primarily a measure of physical efficiency and not of cardiac function. Scott (1921) applied the Schneider rating to the classification of a number of aviators with successful results but pointed out that two conditions, bradycardia and psychical disturbance might vitiate the test by giving a lower rating than the candidate deserved. The daily variations in this test were also investigated by Schneider & Truesdell (1923). Williams (1923) was unable to correlate Schneider ratings with those given by a jump test for physical efficiency and Damez et al (1926) found no acceptable correlation between the results given by the Schneider and the Crampton tests in a series of athletic and non-athletic girls. In their opinion the two indices in Schneider's test which were of most importance as criteria of physical efficiency were the pulse rate while lying down

and the increase in pulse rate after the standard exercise. Lee & Van Buskirk (1923) did not find Schneider's or Crampton's tests useful in measuring fatigue and Seham & Egerer-Seham (1923) came to the conclusion that neither Crampton's, Schneider's nor Barringer's tests were of any value in the diagnosis of physical fitness or cardiovascular ability in children with tuberculosis or heart-disease. Very elaborate multiple tests were devised by McCurdy & Larson (1935a and b) and by Nylin (1937).

Recently the war has led to a revival of interest in cardiac tolerance tests, especially in connexion with the problem of effort syndrome. The Horder Committee (1940) recommended a revised step test (mounting a 15 inch step twenty times in 60 seconds) the pulse rate being taken immediately afterwards over 15 seconds and again one minute after the end of the exercise. This test was found useful by Chamberlain (1941) but according to Parkinson (1941) it is of little value in the diagnosis of effort syndrome. A new pulse-rate test of fitness for strenuous exertion was published in 1942 by Johnson, Brouha & Darling, the index of fitness being given by:

$$\frac{\text{Duration of a standard exhausting exercise in seconds} \times 100}{2 \times \text{sum of pulse rates from } 1-1\frac{1}{2}, 2-2\frac{1}{2} \text{ and } 4-4\frac{1}{2} \text{ mins. in recovery.}}$$

A score below 40 indicated poor fitness. Later, a slightly modified version was employed (Brouha, Graybiel & Heath, 1943)

and Gallagher & Brouha (1943 and 1944) adapted the formula to moderate exercise using a step test of four minutes' duration. The exclusion of the pre-exercise pulse rate from the formula was claimed to minimise psychological effects. The index was said to improve with training and to be depressed during any intercurrent infection. A somewhat similar step test was used by Johnson & Robinson (1943) in the selection of men for physical work in hot climates. In Germany, Müller (1943) introduced a new pulse quotient involving oxygen intake as well as pulse rate. An interesting result was obtained by Taylor (1944), who found that during an exhausting treadmill test on 30 subjects the curves of cardiac acceleration and the heart rate at the mid-point of the exercise were highly correlated with the endurance time. In an attempt to minimise variations of response in any given subject, Foltz, Ivy & Barborka (1942) advocated the use of double work periods with an intervening rest period. A useful general review of fitness tests was given by Taylor & Brozek (1944). In their opinion the heart rate after a standard amount of work is probably the most useful single criterion of fitness during convalescence.

On the negative side, the Schneider rating has again been attacked, particularly by Fiel, Petti & Park (1943) who found that it may fail to differentiate heart patients from normals; and by Taylor & Brown (1944) who showed that though

valid when selected fitness groups were compared, the test fell short of requirements when applied to individuals. Similar results were obtained by Barrow & Ouer (1943) as regards its correlation with electrocardiographic changes. The validity of exercise tests in general has been questioned recently by Levy, Stroud & White (1943), and by the National Research Council sub-committee on Cardiovascular Diseases (1943). Thus again tests which appeared effective in the laboratory have proved of doubtful value in the wider field of clinical application.

For the purposes of the present research it was finally decided that some form of step test would prove most suitable. Hunt & Pembrey (1921) summed up the advantages of such tests as follows:

- (i) It is a form of exercise to which all are accustomed.
- (ii) Although a heavy man walking upstairs does more work than a light man, what we really want to know and compare is the capacity for moving the body from place to place, especially in pathological cases; i.e. to know the relative efficiencies of men as walking machines. Peabody & Sturgis (1922) and White (1937) also stressed the importance of using an exercise which enters into the routine of daily life.

As it was proposed to carry out the present test on cardiac patients as well as on normal subjects a very

light exercise was desirable, and some modification of Master & Oppenheimer's two step test seemed to offer the best solution. A further advantage of such a test is that both the rate of working and the duration of exercise can readily be controlled, a necessity in the case of a brief light exercise. In addition, though there had been a fair amount of previous work on the heart rate during moderate and strenuous exertion, there appeared to be very little on the course of cardiac acceleration during a brief light effort such as walking up an ordinary short flight of stairs. In the present instance a framework consisting of two steps each exactly ten inches high was used; these are well shown in the foreground of Fig. 6. It was decided that the subject should step five times up and down these, bringing both feet together at the top and descending backwards on the same side of the steps. This procedure is simpler than that of Master & Oppenheimer who made their subjects step up one side, down the other, and then walk round the steps to the starting point. The rate of climb must also be controlled and this was achieved by making the subject step in time with a metronome beating 96 to the minute. These precautions ensured a reasonable uniformity in the performance of the test, but as it was designed for clinical use, no extreme precautions were taken to obtain a true basal initial rate, and slight latitude was allowed

in the carrying out of the exercise.

The complete procedure for the standard tolerance test was as follows:

The metronome was started and the subject had a brief practice in ascending the steps. The chest electrodes and stethograph were then applied and the subject sat down on a chair eighteen inches high and relaxed as fully as possible. At the end of three minutes a tracing was run for 30 seconds, the subject remaining relaxed. The drum was then stopped and a further two minutes' rest allowed, giving a little over five minutes relaxation in all.

Master & Oppenheimer (1929) and Master (1934) stated that a steady resting rate is usually reached in from 3 - 5 minutes under such conditions. The drum was then started and the resting heart-rate recorded for 30 to 40 seconds. The subject was then told to begin the exercise and the exact moment when he left the chair was signalled on the drum. The subject rose to his feet, stepped five times up and down the steps and then sat down again and relaxed, the moment of sitting down also being signalled on the drum. The heart rate was recorded continuously during the exercise and for 3 - 4 minutes afterwards. The average duration of the exercise from rising to sitting again was just over 20 seconds. It will thus be seen that this standard exercise was both lighter and of much shorter duration than that of the usual type of cardiac tolerance test.

SUMMARY.

A general review of the previous work on exercise tolerance tests is given. The advantages and disadvantages of the various types of test are discussed and reasons given for the selection of a step test as the standard exercise for the present investigation.

A detailed account of the procedure for this standard test is included. In brief, the exercise chosen consists in ascending and descending two steps each ten inches high five times in approximately twenty seconds. The exercise begins and ends in the sitting posture.

"A variety of perplexing circumstances soon convinced me of the necessity there was, correctly to ascertain the various conditions of the healthy pulse, particularly as regarding muscular exertion, diet, etc. This knowledge, however, I found was not to be obtained in a short time, nor without considerable labour."

Robert Knox, (1815) p.52.

CHAPTER 6.

THE EFFECT OF THE STANDARD EXERCISE ON THE HEART
RATE OF NORMAL SUBJECTS.

Series A and B.

In order to obtain data on the behaviour of the heart rate in normal subjects during the simple step test described above, the test was performed by 100 subjects, 75 men and 25 women. To determine whether there were any significant sex differences in the behaviour of the heart rate the two groups were analysed separately. Before each subject performed the test, various measurements were taken, including weight, height, chest girth, blood pressure, etc., together with a short medical history with special reference to any cardiac complaints. All the data were entered on a standard card as shown in Fig. 11 in order to assist the statistical work.

In Series A seventy-five male medical students acted as subjects. They were in good health but not in training. Their ages, in nearly every case, lay between 18 and 22 years.

In Series B twenty-five female medical students acted as subjects. Their age range was between 18 and 25 years.

RESULTS.

The results from Series A and B for the various heart rate indices previously described are summarised in Table 1, and will now be discussed in detail.

DISCUSSION OF INDICES OBTAINED IN SERIES A AND B.

(1) Initial Rate.

(Heart rate in 10 second period immediately preceding exercise).

The literature on the sitting pulse rate is somewhat scanty as resting pulse rates are generally taken in the recumbent posture, but Table 2 gives a summary of the main results obtained by previous authors. From this it would appear that the mean rates in the present series (85.75/min. for men and 90.35/min. for women) are rather higher than might be expected, though the sex-difference is in general agreement with the authors cited in Table 2 and with the work of Lombard & Cope (1928). These high initial rates are probably the result of psychological factors, as the subjects knew that the exercise was about to commence, though care was taken to see that the recording apparatus was not visible to them. This is borne out by the fact that the average heart rate taken after the first three minutes of rest (early resting rate) is 81.5/min. in the men and 84.4/min. in the women; while after a further two minutes of rest the rates have risen to 85.75 and 90.35/min.

respectively, immediately before exercise. This increase of heart rate with emotion has of course been noted ever since accurate counts were possible. For example, it is mentioned by Bryan Robinson (1732) and by Falconer (1796), and Rucco (1827) gives an amusing account of it as follows.. ".....and in fact the beating of the pulse varies in proportion as the exercise of the intellectual faculties is increased or diminished; because when their exercise is raised to a certain point, which is frequently to be seen in extemporaneous poets, and in orators of great eloquence and superior wit, the pulse then beats quickly, and its pulsations are more energetic and agitated." In more humble circumstances the emotional increase has always been a disturbing factor in cardiac tolerance tests. Billings & Shepard (1910) found that while conscious attention invariably increased the heart rate, sensory attention may decrease it; and Schwab (1927) went so far as to say that emotion was the most common accelerator of the heart, while Whitehorn, Kaufman & Thomas (1935) showed that the heart responds with a brief acceleration to even the most fleeting emotions. According to Boas & Goldschmidt (1932) the emotional acceleration is slightly greater in women than in men and this is true for the present series, the average increase being approximately 4 beats/min. for the men and 6 for the women. As in the present case,

Ellis (1932) found that the resting pulse rates of his subjects were higher than might have been expected, owing to excitement. Quantitative data were given by Addis (1922) who found that excitement due to the mere act of counting the pulse might raise the pulse rates of normal subjects by 12 beats per minute; and by Peabody & Sturgis (1922) who found that the heart rates in the 20 seconds before an exercise test began were from 5 to 9 beats per minute higher than during the previous 5 minutes of rest. These increases agree well with those obtained in the present investigation. Robinson (1939) observed that the anticipatory increase in heart rate immediately before moderate work is variable and has no constant relation to age. Much work has been done on the immediate cause of this emotional acceleration. Schneider (1936) pointed out the curious anomaly that though mental excitement has so little effect on metabolism it has such a large effect on the pulse rate. In his opinion the liberation of adrenaline plays a part in the acceleration, and McDowall (1938) is in agreement with this. Experiments on the cat also support this view (Cannon & Britton, 1927, and Britton, Hinson & Hall, 1930). Direct nervous control of the heart rate is also important (Bond, 1943), and it would appear from the experiments of Brouha & Nowak (1939 a and b) on

dogs that the vagus plays a more important part than the sympathetic in bringing about the emotional acceleration. In support of this also may be cited the experiments of Fleisch (1933) who found that the increased rate could be immediately lowered in man by bringing the oculo-cardiac or Valsalva reflexes into action. That the increased heart rate in excitement is accompanied by an increased cardiac output was shown by Grollman (1929) in man. General reviews of the effects of emotion on the heart rate were given by Dunbar (1935) and Darrow (1942).

Since it would appear to be impossible to eliminate emotional factors, especially in clinical work, the high initial heart rates must simply be accepted as inevitable. As was pointed out by Burns (1939) they are really part of the normal preparation of the body for exercise and are necessary for maximum efficiency. The fact that they are unusually high in the present series is merely because the method of recording allows accurate counting of the rate right up to the moment exercise begins. Their influence on the various indices will be discussed later.

No data seem to be available concerning the variability of the sitting pulse rate except those of Hill, Magee & Major (1937) who found the coefficient of variation to be 10.5% in 29 male students; but coefficients for the

recumbent resting pulse rate are in good agreement with the figure of 16.1% obtained in the present series for the sitting rate. Thus Addis (1922) found the recumbent rate in men to have a coefficient of variation of 16%, and Jackson (1927) gave 15.1% in a series of 1600 male students. In women, Jackson (1929) found the coefficient to be 11.5%, though Sutliff & Holt (1925) had previously found the basal pulse rate to be more variable in females than in males. For the standing pulse rate, Franke (1928) gave the coefficient of variation as 15.9% for men, while Griffith et al. (1929) found it to be 8.5% for men and 8.8% for women.

Various attempts have been made to correlate the resting pulse rate with the body weight, usually without success. In the present instance, there is no significant correlation, the coefficient for series A and B, taken together, being - 0.069 with a standard error of 0.1025. This is in agreement with the results of Harris & Benedict (1919), Boas & Goldschmidt (1932) and Hill, Magee & Major (1937).

(2) The Maximum Rate.

(Highest rate reached in a 5-second period during the exercise).

It is difficult to find comparable results in the literature for the maximum rate taken during exercise, as of course the rate of working and the duration of the work must be taken into account. In the present series a subject of

average weight works at the rate of approximately 400 to 500 kg.m/minute while performing the standard exercise. Bowen (1903) measuring the pulse rate in 10 second periods during exercise found a maximum rate of 115/minute after 20 seconds when work was performed at the rate of 354kg.m/minute; while Paterson (1928) found a maximum of 140/minute in a period of 15 seconds while work was carried out at 650 kg.m/minute. The order of magnitude of the maximum rate in these results is in quite good agreement with the mean figures for the present series, which are 130/minute for men and 132.6/minute for women. It is interesting to note however, that a rate as high as 130/minute is reached in healthy young men and women performing what is after all a very mild exercise. The slight sex difference in maximum rate is in agreement with the results of Taylor (1941) for more severe exercise. An important feature of the maximum rate is that it is the least variable of all the indices chosen, its coefficient of variation being only 9.9% in men and 10.3% in women. Thus it would appear to be one of the most suitable indices for the determination of cardiac tolerance.

Relationship of the Maximum Rate to the Initial Rate.

Contrary to the results obtained by Boas (1931b) for exhausting exercise, the maximum rates in my series are very significantly correlated with the initial rates; the

coefficient of correlation in Series A being +0.69 with a standard error of 0.116, and in Series B +0.695 with an standard error of 0.204. This means of course that if the initial heart rate is high the maximum rate reached tends also to be high. It has already been shown that high initial rates in the present series are probably due to psychological causes.

It does not follow, however, that emotion simply raises the levels of the heart rates evenly throughout the test. Detailed examination of the results shows that where the initial rate is high the absolute increase in heart rate produced by the exercise is generally small. For example, if the 75 subjects in Series A are divided into those with an initial rate below and those with an initial rate above 85 per minute the following results are obtained:-

With initial rate below 85/min. the increase with exercise averages 48.3 beats per minute.

With initial rate above 85/min. the increase with exercise averages 40.15 beats per minute.

This diminished increase with the higher initial rates is particularly noticeable in those subjects whose resting rate immediately before exercise shows a considerable increase over their resting rate taken two minutes earlier, and those are presumably the subjects in whom the emotional

element is most marked. It would thus appear that during the exercise the emotional increase is lessened and that some factor comes into play to prevent the occurrence of an excessively high maximum rate. This phenomenon does not appear to have been described before in connection with very mild exercise, though it has been noted during and immediately following exhausting exercise. Thus Boas (1931) recording the heart rate during exhausting exercise in boys, found that the lower the initial heart rate the greater was the increment in rate, and a similar observation on dogs was made by Essex, Herrick, Baldes & Mann (1939). Cotton, Rapport & Lewis (1917a) obtained similar results as regards the relationship between the initial rates and the maximum rates recorded immediately after maximal exercise in 'irritable heart' patients and controls. They explained the phenomenon on the assumption that: "the circulation is capable at a given moment of a certain response to a given effort, and it appears to be a matter of indifference whether this response has been called forth to some extent before the chief stimulation has been applied providing it has been called forth through similar channels." In the present series, if emotion be considered to play the part of the introductory stimulus before the chief stimulus is applied, the results are in accordance with this theory, although the exercise is very

far short of maximal. Hunt & Pembrey (1921) pointed out that if the speed limit of the ventricles in the normally acting heart is assumed to be about 180/minute, then the slower the pulse rate at rest the greater is the possible variation available for increased muscular work, hence the advantage to the athlete of a slow pulse at rest. Thus one would expect a greater increase of pulse rate over a low initial rate than over a high one when the exercise is maximal, as presumably the subjects with low initial rates will be able to carry out more work before becoming exhausted. This theory of what might be called the 'frequency reserve' does not however operate to the same extent in the case of a standard mild exercise of short duration, and it must be emphasised that in the experiments of Cotton et al. the amount of exercise performed differed for each subject, the constant factor being the degree of distress produced. This accounts for their finding that in the 'irritable heart' group the maximum post-exercise rate was actually higher when the initial rate was low than when it was high, an effect which certainly does not occur in the present series.

(3) The Time of Occurrence of the Maximum Rate.

(Time from beginning of exercise to middle of 5 seconds period in which the maximum rate occurs).

In most of the previous work on this subject, it was found that the maximum pulse rate reached during a short exercise occurred at the end of the exercise period. Thus Peabody & Sturgis (1922) counted the heart rate in consecutive periods of 15 seconds during an exercise lasting one minute and nearly always found the maximum rate in the count during the last 15 seconds of exercise. Similar results for more prolonged exercise in man were obtained by Iljin-Kakujeff (1935) and Taylor (1944). In the present series a different result was obtained. The maximum rate, instead of occurring at the end of the 24 seconds of exercise, is reached on the average after 15 seconds of exercise in the men and after 17 seconds in the women. The reasons for this early occurrence of the maximum rate will be fully discussed later, in the section on the general curve of heart rate during the standard exercise.

(4) The Acceleration of the Heart Rate.

(Maximum rate minus rate in 5 second period immediately preceding exercise divided by time to reach maximum rate.)

Various workers have divided the increase of heart rate during exercise into more or less definite stages; for example the 'Initial Acceleration' and 'Persistent Acceleration' of Merklen (1926) and the 'Initial', 'Action' and 'Recovery' accelerations of McDowall (1938). It is difficult to apply these nomenclatures to the changes observed during an exercise which lasts for less than half a minute, but the acceleration discussed here probably corresponds mainly to the 'Initial Acceleration' of the above workers. Incidentally the use of the word 'acceleration' in most of the papers on heart rate is open to question. It has been very generally used to denote merely "change of frequency" instead of in its real sense of "rate of change of frequency," and it should be noted that the present index is the first quantitative measure of the true acceleration expressed as beats per minute per second (Knox, 1940). In the present series the mean acceleration is of the order of 2.5 to 3 beats per minute every second until the maximum rate is reached. This index is, however, subject to great variations in different individuals, the coefficient of variation being 28.6%

in Series A and 36.8% in Series B. This is of course due, at any rate in part, to the number of variable factors which enter into its calculation; and particularly to the fact that the time of occurrence of the maximum rate can only be estimated to the nearest five seconds.

A very striking feature of the cardiac acceleration which has long been known is its extremely short latent period. This was noted by MacWilliam (1893) and Grunbaum & Amson (1901). It was confirmed by Bowen (1903) who found that the acceleration had begun by the second beat; and Buchanan (1909), using a capillary electrometer, found that even in very light exercise such as clenching the fist the first diastole of the heart after the exercise began was shortened. Boas (1931b) using the cardiometer obtained the same result, which is also clearly demonstrated in the present tracings. (See Fig. 12).

It will be seen from the graphs of mean heart rate during the step-test (Figs. 13 and 14) that the course of the acceleration up to the time of occurrence of the maximum rate is not an even one. Up to 7.5 seconds from the beginning of exercise the graph is practically a straight line, indicating a steady acceleration, but after this point the acceleration rapidly falls off until the maximum is reached. This is particularly noticeable in the

Series B graph (Fig. 14). The exact figures for the mean acceleration are 3.0 beats/min./sec. for the men and 2.61 for the women (see Table 1). The lower acceleration in the women is no doubt due largely to the fact that when exercise began their heart rate was already accelerated by emotional factors to a greater extent than in the men, as was pointed out above. Using a more severe exercise Metheny, Brouha, Johnson & Forbes (1942) found the cardiac acceleration to be greater in women than in men.

There was no significant correlation between the body weight of the present subjects and the acceleration of the heart rate (Series A + B). The coefficient of correlation $r = +0.109$ with a standard error of 0.1025.

The deceleration of the heart rate after the exercise will be discussed under the heading "The Return to Normal".

(5) Number of Extra Beats induced by the Exercise.

(Number of beats in the 30 seconds after starting exercise minus half the initial rate).

The possibility that some information might be obtained by calculating the number of extra heart beats produced by the exercise in a given period was next considered, and the point which then arose was which period of time to choose. As the index required was the number of extra beats, it appeared logical to calculate this number in the period from the beginning of exercise to the time of return to normal, as this period would presumably include all the extra beats. This was accordingly done, but the results were disappointing owing to their extreme variability. In Series A the mean was 20.2 beats with a coefficient of variation of 37.8%; and in Series B the mean was 21.5 beats, the coefficient of variation being 27.5%. In addition, the counting of the beats over such a long period was extremely laborious and so a simplified version of this index was introduced as follows. The number of extra beats in a standard time was calculated, the time chosen being the 30 seconds following the beginning of exercise, which included the whole of the exercise period in all cases. Actually, not many extra beats were lost by this procedure, the means being 17.0

beats in Series A and 16.4 beats in Series B, but the coefficients of variation were now 28.6% for Series A and 20.8% for Series B, a considerable reduction. This index is of no great value in the present series of healthy subjects, but it was thought that it might prove of interest in connexion with the cardiac patients to be described later. There was no significant correlation between the body weight and the number of extra beats induced by the exercise.

(6) Actual Increase in Beats per Minute caused by the Exercise.

(Obtained by subtracting the Initial Rate from the Maximum Rate).

The relationship of this index to the initial rate was dealt with under the heading of "Maximum Rate", when it was shown that the absolute increase in heart rate tended to vary inversely as the initial rate. Apart from some figures given by Bowen (1903) for very light exercise (tapping a telegraph key) and by Boas (1931b) for exhausting exercise, the only results calculated from observations actually taken during exercise are those of Peabody & Sturgis (1922). They found an average increase of 31 beats per minute, with extreme variations between 23 and 46 beats/min. during an ascent of 60 steps each 18 cm. high in one minute. Though little has been done on the actual increase during exercise, there are numerous records of the difference between the initial rate and the immediate post-exercise rate. As it will be shown later that the post-exercise rate is highly correlated with the maximum rate reached during exercise, it is permissible to include some of these results here.

Dana (1919) concluded that "one can judge accurately enough in most cases as to the effect of work upon the heart rate by observing the degree of acceleration of the pulse after 20 - 40 hops, and the promptness with which the heart action again becomes quiet." Hunt & Pembrey (1921)

while attempting to differentiate the response to exercise of trained and untrained men, showed that the actual increase in pulse rate was as good a criterion as the time of return to normal. Flack & Bowdler (1920) using an exercise test comparable with the present one, (five climbs on to a stool 18 inches high in 15 seconds), concluded that if the immediate post-exercise rate exceeded the initial rate by more than 25 beats/minute the subject's cardiovascular system should be regarded as suspect. Using the same exercise however, Schneider & Truesdell (1922a) were unable to demonstrate any difference between an unselected group of aviators and a group specially selected for their physical fitness, as indicated by the increase in pulse rate alone. The superiority of the fit group was most clearly shown by its lower standing rate, its smaller reclining to standing increase, and a more rapid return to the normal standing rate after exercise. Cripps (1924), employing the same test on women, obtained results comparable with those of Flack & Bowdler. In the present series the average increase was 44.4 beats/minute in Series A and 42.3 in Series B, the coefficients of variation being 22.8% and 21.4% respectively. The smaller increase in the women (Series B) is again accounted for by their higher initial rate. The variability of the index here is much less than in the series described by Fränke (1928). Using a

knee-bending exercise, he found the coefficient of variation of the increase of pulse rate to be as high as 45%. Hill, Magee & Major (1937) observed the rise of pulse rate after two maximum pulls on a dynamometer and found that the absolute rise in rate was the same in individuals with slow, medium and fast initial rates. This is in marked contrast to the present results, possibly due to the different type of exercise employed.

The present results differ from that of the actual increase, in that the percentage increase is higher for the slower initial rates. This is because, as was pointed out, the slower initial rates have a greater actual increase in rate of work and consequently have a still greater percentage increase. This difficulty was also experienced by Hill, Magee & Major (1937). The effect of emotional factors on the percentage increase in pulse rate was indicated by Gillespie (1924), who found that the response to muscular work was much greater than that to mental work; and that the response to combined mental and muscular work was greater than to either alone. Kerkian

(7) Percentage Increase on Initial Rate.

$$\left(\frac{\text{Max. Rate minus Initial Rate.}}{\text{Initial Rate}} \times 100 \right)$$

According to Merklen (1926), this index is an improvement on the 'absolute increase' because it enables a better comparison to be made between the reactions of different subjects to the same exercise. This would at first sight appear to be logical, as the percentage increase takes more account of the initial rate. In practice, however, its coefficient of variation is considerably greater than that of the actual increase, as may be seen from Table 1. This is because, as was pointed out earlier, the low initial rates have a greater actual increase than the high ones and consequently have a still greater percentage increase. This difficulty was also experienced by Hill, Magee & Major (1937). The effect of emotional factors on the percentage increase in pulse rate was discussed by Gillespie (1924), who found that the response to muscular work was much greater than that to mental work; and that the response to combined mental and muscular work was greater than to either alone. Merklen (1926) invented a further index which he called the 'coefficient of acceleration', obtained by dividing the sum of the initial rate and the maximum rate by the difference

between them, i.e. by the 'actual increase'. This index does not appear to have any advantages over the more usual ones, however.

and it was therefore necessary to include
in the present work for purposes of comparison
able time had to be allowed for finding the
very end commencing the timing of the pulse, the
not started until 5 seconds after the start of
The average results were 91.7 minutes/minute in
and 96.2 in the women (see Table 2.)

In order to determine whether the time taken
during exercise could be decided from the
rate with any degree of accuracy, the
correlation between these factors was

with the following results:-

men) - coeff. of correlation 0.15.
Standard Error 0.115
women) - coeff. of correlation 0.26.
Standard Error 0.208.

(8) The Post-Exercise Rate.

(Rate in beats per minute counted over a 30 second period, beginning 5 seconds after the end of exercise).

In most of the previous work on the rise of the pulse rate with exercise the only criterion available was the rate counted by palpation as soon as possible after the exercise ended, and it was therefore necessary to include such an index in the present work for purposes of comparison. As a reasonable time had to be allowed for finding the radial artery and commencing the timing of the pulse, the count was not started until 5 seconds after the end of exercise. The average results were 91.7 beats/minute in the men, and 98.2 in the women (see Table 1.)

In order to determine whether the true maximum rate occurring during exercise could be deduced from the post-exercise rate with any degree of accuracy, the statistical correlation between these indices was calculated with the following results:-

Series A (men) - coeff. of correlation +0.83.
Standard Error 0.116.

Series B (women) - coeff. of correlation +0.30.
Standard Error 0.208.

These indices therefore are very significantly correlated with one another and thus the post-exercise rate, which requires no apparatus and can be obtained by palpation

gives a fairly reliable indication of the maximum rate actually reached during the exercise.

The regression equations connecting the two indices are given below:

Series A (Men).

Maximum Rate = $0.72 \text{ post-exercise rate} + 64.$

Series B (Women).

Maximum Rate = $0.71 \text{ post-exercise rate} + 63.$

The excellent agreement between the regression equations for these two entirely separate series makes it unnecessary to calculate the regression equation for a combined series of men and women. By the use of these equations the probable maximum rate during the exercise may be calculated from the post-exercise rate alone.

Cotton & Dill (1935) using a modified Boas' cardiometer recorded the heart rate in four periods of ten seconds each, two immediately preceding and two immediately following the end of the exercise. The exercise consisted of walking and running on a flat treadmill and was continued long enough for a steady state to be reached. They came to the conclusion that the heart rate during exercise may be predicted from that recorded in the 10 seconds period following exercise, with an error whose Standard Deviation is rather less than 3%. They also found that the heart rate fell very little during the

first ten seconds after the exercise, and for the next ten seconds only about 6%. It must be noted that the conditions differed from those of the present series in that their exercise was prolonged until a steady state was reached. This, no doubt, accounts for the slow deceleration after the exercise, in contrast to the rapid drop observed in my subjects. Gillespie, Gibson & Murray (1925) stated that while the pulse rate immediately after exercise was a fairly reliable relative indication of the rate of the pulse during the preceding work, it was impossible to draw inferences therefrom as to the absolute rate of the heart during exercise. Paterson (1928) recording the heart rate during bicycling by means of a wrist tambour, found that the values of the pulse rate during exercise were much higher than one would expect from values taken even immediately after exercise. This is upheld by the present results.

It should be remembered that in most of the previous experimental work this post-exercise rate has been taken as the maximum rate, and from the foregoing it would appear that the results thus obtained should be applicable in large measure to the present series. For example, as mentioned above Flack & Bowdler (1920) using an exercise very similar to the present one concluded that if the increase of the post-exercise rate over the initial rate was greater than 25 beats/minute, the cardiovascular system was

faulty. It is of interest, therefore, to calculate this increase in the present series. In Series A it averages 6 beats per minute and in Series B, 8 beats/minute and thus the averages of both series come well within the limit set by Flack & Bowdler, and this in spite of the very high true maximum rate.

As regards the actual value reached by the post-exercise rate, Parkinson (1917a) found that when normal subjects climbed 25 steps at a moderate walking pace the rate in the 30 seconds immediately following exercise was 93 per minute. Both the exercise used and the result are very comparable to the present ones. Elbel & Green (1946) using a single step 20 inches high and a stepping rate of 24/minute, found that the immediate post-exercise rate averaged 103.6 beats/minute when the exercise lasted 30 seconds. That very high post-exercise rates (up to 172 per minute) may be found after severe exercise was shown by Hill & Flack (1909).

The post-exercise rate is of course subject to considerable variations, its coefficient of variation being 16.15% in Series A and 15.8% in Series B. Cotton, Rapport & Lewis (1917a) and Lythgoe & Pereira (1925) have actually observed slight increases in the pulse rate after the end of exercise instead of the usual immediate fall, and this result very occasionally appears in the present tracings.

Where present, it will of course tend to produce an abnormal post-exercise rate.

It will be seen from Table 1 that the post-exercise rate is higher in the women than in the men students, and this goes with the slower descent of the pulse rate curve in the former as shown in Fig. 14. Emotional factors are probably responsible for the difference and in this connection it is interesting to note that Gillespie (1924) found that mental work alone raised the pulse rate more in women than in men, though the blood pressure was raised much more in the men than in the women.

The value of the post-exercise rate was emphasized by Lewis (1917) who held that in D.A.H. and V.D.H. patients "the average height to which the pulse rate is raised at the cessation of effort may be taken therefore to gauge the degree of distress produced when an additional objective sign is desired". Dana (1919) was also of this opinion. Hartwell & Tweedy (1913) and Hunt & Pembrey (1921) showed that after a given exercise the post-exercise rate is higher in untrained than in trained subjects, as might be expected.

(9) The Return to Normal.

The time required by the pulse rate to return to normal after effort has been very widely used as an index of physical condition; but, as with all such indices, there is little agreement as to its value. Cook & Pembrey (1912) found the return to normal to be their best criterion of good cardiovascular accommodation and training in men, and Hartwell & Tweedy (1913) showed that it was considerably shorter in athletic girls than in non-athletic girls. More detailed investigation of the effect of training was carried out by Robinson, Edwards & Dill (1937) and by Dill & Brouha (1937). They observed that the recovery of the heart-rate took place at the same speed in both trained and untrained during the first half minute, after which the athletes' heart rate fell more rapidly. This was, however, later denied by Knehr, Dill & Neufeld (1942) who found that training did not materially alter the course of the recovery heart rate. Hill, Magee & Major (1937) found the time of return to normal to be the most constant feature of their dynamometer test. The physical fitness tests devised by Greifer (1939) and Hardy, Clarke & Brouha (1943) depended mainly on the time of return to the resting heart rate. As regards its value in cardiac abnormalities, Lewis (1917) showed that the time of decline of the pulse

rate in a series of D.A.H. patients who showed symptoms after exercise was five times longer than the corresponding time in a series of patients showing no symptoms. Later, Burns (1939) laid considerable stress on the value of the return to normal, while the Horder Committee (1940) recommended its inclusion in their standard exercise tolerance test. On the other hand, Hunt & Pembrey (1921) and Merklen (1926) have pointed out that an accurate determination of the return to normal presents considerable practical difficulties. The main sources of error would appear to be as follows:

- (1) The resting rate is itself by no means an absolute constant.
- (2) The pulse rate after exercise, instead of returning smoothly to the original rate, often stops short a little way above the resting level and may not return to it for a very prolonged period (Deane, 1910 and Benedict & Cathcart, 1913). When in this state, a very slight exertion may cause the pulse rate to 'click' suddenly down to its original resting rate (personal communication from Professor Cathcart). In this connexion it is of interest that Edgcomb & Bain (1899) found that very gentle resistance exercises may actually lower the pulse rate. Mosler (1912) also found that the return to normal seemed to occur in a series of steps rather than in a smooth curve.

(3) A quick return to normal or subnormal may be sustained only for a few seconds and the pulse rate may then rise and not return again to normal for several minutes. A period of subnormal pulse rate after exercise was described by Chailly-Bert & Langlois (1921). This so-called 'post-exercise trough' and the subsequent 'secondary rise' were investigated by Klyver, Huang & Shafer (1927) who regarded them as merely the beginning of the normal fluctuations of the resting pulse rate, to be accounted for by the causes which give rise to these normal fluctuations. Cotton (1928) while offering no explanation of the 'trough' threw doubt on the results of Klyver et al. on the grounds that their method of counting the pulse was neither continuous nor as accurate as it might have been. Several of the tracings in my series show evidence of a post-exercise trough and a possible explanation will be offered in Chapter 8. Because of these fluctuations Meakins & Gunson (1917) pointed out the importance of counting the pulse rate in periods which are as short as possible. They counted the rate after exercise in 6 second periods.

As described in Chapter 4, the return to normal in Series A and B was calculated by a new method rendered possible by the continuous recording, thus:

The exact space occupied by the last ten heart beats before exercise began was marked off on the edge of a piece of paper. This paper was then slid along the tracing until the next set of ten beats which would occupy this space was found. The time from the end of the exercise to the first of these ten beats was taken as the time of return to normal. It was at first feared that the onset of sinus arrhythmia in some of the tracings might upset this count, but it was found that this was not so. This micro-method has the advantage of great exactness, but the results show considerable variations from individual to individual. In Series A (men) the average time of return to normal was 21.4 seconds with a Standard Deviation of 16.42 and a Coefficient of Variation of 77%. In Series B (women) the average time was 25.4 seconds with a Standard Deviation of 9.35 and a Coefficient of Variation of 36.8%. Using a comparable exercise and counting the pulse in 5 second periods Treadgold (1930) found the average return to normal in healthy men to be about 15 seconds, and Flack & Bowdler (1920) said that it should occur within 30 seconds of the end of their exercise; results which are fairly close to those of the present series. It is interesting to note that Cripps (1924) found the return to normal to take longer in women than in men, but the differences between the results in Series A and B are hardly great enough to be significant in this respect, though a

more prolonged time in the case of the women might be expected from their higher post-exercise rate and slower decline (see Fig. 14).

The real drawback however to the use of the return to normal obtained in this way as an index is its extreme variability in normal individuals. The coefficient of variation, especially in Series A, is very high indeed and for this reason the index has seldom been so calculated in the subsequent special series. No significant correlations were found between the time of return to normal and the body weight or between the time of return to normal and the haemoglobin percentage of the subjects.

The above-mentioned variability of the return to normal is a well-known phenomenon and has been described by many authors. It is probably that this does not represent the phenomenon of the so-called "post-exercise trough" in the haemoglobin described by Lowsley (1912) and investigated by Elysee, Hugg & Shafer (1927) and by Cotton (1928), as the time relations are quite different.

(10) The Rate 5 minutes after exercise.

(Counted over 30 seconds beginning 5 minutes after end of exercise).

As the average time of return to the initial rate was less than 30 seconds it was to be expected that this index would be at least as low as the initial rate. The actual results, 80.3 beats/minute in the men and 82.2 in the women, show that the heart rate has by this time fallen considerably below the initial rate (counted in the 10 seconds immediately preceding exercise) and is even slightly lower than the early resting rate taken two minutes before exercise began; in fact, it is the lowest of all the heart-rate indices calculated. The main reason for this is almost certainly a psychological one. The test having been safely completed and the tension relaxed, both the exercise and emotional accelerations have passed off. It is probable that this low rate bears little relation to the so-called "post-exercise trough" in the heart-rate described by Lowsley (1911) and investigated by Klyver, Huang & Shafer (1927) and by Cotton (1928), as the time relations are quite different.

SUMMARY .

Using 75 men and 25 women as subjects, the effect of the standard exercise on the following heart rate indices is discussed.

- (1) Initial rate.
- (2) Maximum rate.
- (3) The time of occurrence of the maximum rate.
- (4) The acceleration of the heart rate.
- (5) The number of extra heart beats induced by the exercise.
- (6) The actual increase in heart rate.
- (7) The percentage increase in heart rate.
- (8) The post exercise rate.
- (9) The return to normal.
- (10) The heart rate five minutes after exercise.

The mean values and variability of these indices are given and the results discussed in relation to those of other workers.

The maximum rate during exercise is found to be significantly correlated with both the initial rate and the post exercise rate.

"At all times it must have been observed, that muscular exertion, almost of every kind, but more especially violent exercise, increased greatly the powers of the heart and arterial system; but that this extended even to the slightest muscular motion, such, for example, as is made use of during a change of posture, does not appear to have been suspected, or if so, its importance has been greatly overlooked."

Robert Knox (1915). p. 165.

CHAPTER 7.

THE EFFECT OF THE POSTURAL AND EXERCISE COMPONENTS OF
THE STEP-TEST ON THE HEART RATE.

In order to obtain a general picture of the average changes in heart rate during the standard exercise the mean rates in each five second interval were calculated. Series A (75 men) and Series B (25 women) were treated separately and the results are shown in Figs. 13 and 14. The mean rates are plotted in the centre of the 5 second interval in which they occur. It will be seen that in both series the heart rate increases linearly up to 7.5 seconds after the beginning of exercise, after which the acceleration falls off. Following the maximum, the rate falls slightly although the exercise is still in progress. After exercise ends the rate falls much more rapidly in Series A than in Series B. These are merely general expressions of results which have been discussed in detail in the sections on the various heart rate indices.

In the section on "The Time of Occurrence of the Maximum Rate" it was pointed out that the results of most previous workers indicated that the maximum rate occurs at the end of a short exercise. That this is not the case in the present series is clearly shown in the graphs.

An explanation of this decrease in rate during the exercise was then sought. It seemed possible that in a

very short light exercise of the present type, where the rate was counted accurately over very small periods, the effects of posture might appear in the record and prove the complicating factor. The exercise was begun from the sitting posture and standing erect might augment the heart rate during the first half of the exercise. This postural increase might pass off as circulatory adjustments were made, independent of the actual stepping; thus accounting for the decrease in rate during the last five seconds of exercise.

This theory was tested on a series of seven male students who were well accustomed to the apparatus and in whom the psychological increase was minimal. This presumably accounts for their low maximum rates.

The subjects first sat down and rested quietly until the heart rate was steady, then at a given signal they stood up and remained standing quietly, the heart rate being recorded in the usual way. They then sat down again and rested for 15 minutes, at the end of which they performed the standard exercise starting from the sitting posture. A further 15 minutes rest followed to ensure that the heart rate had returned to the resting level. The subjects then stood quietly for 5 minutes and then repeated the stepping exercise from the standing posture. Graphs of the mean heart rates in 5 second periods during the three tests are

shown in Fig. 15. It will be seen that the curve for the sitting - exercise - sitting test (curve B) shows the typical decrease in rate during the last 5 seconds of exercise, whereas the standing - exercise - standing curve (curve A) continues to rise till the end of exercise. The sitting - standing postural curve (curve C) shows an increase to a maximum followed by a decrease to a resting level about 7 beats/minute higher than the sitting resting rate. As might be expected from the postural theory the curve of acceleration of the heart rate during the first ten seconds of exercise is steeper in the sitting - exercise - sitting test than in the standing - exercise - standing one. It would thus appear that the postural increase and the exercise increase are to a certain extent additive. If this is so, it should be possible to construct a curve for the exercise component alone by subtracting the heart rates in each 5 second period of the sitting - standing test from those in the corresponding periods of the sitting - standing - exercise test, i.e. by subtracting curve C from curve B, Fig. 15. If the components are additive we should expect the resultant curve to rise steadily to a maximum at the end of exercise as in curve A (standing - exercise - standing). The resultant curve is shown in Fig. 16, and follows the general trend expected from the theory. In addition it is to be expected that if the exercise were

continued beyond the standard 20 seconds the rate would increase again following the drop due to the falling off in the postural component. That this is the case will be shown in the section on the effect of duration of exercise, (but see Fig. 22). The present test has thus been shown to combine the effects of posture and of exercise on the heart rate, the total effect being the resultant of the components.

DISCUSSION.

The Decline in Heart Rate during Exercise.

On searching through the records of other workers it was found that a fall in heart rate during exercise did occasionally appear, though it was not stressed by any of them. Thus in experiments on very light work (tapping a telegraph key) by Bowen (1903) the average curve showed the maximum heart rate at $2\frac{1}{2}$ minutes during a 4 minute exercise. Schneider (1916) found that when work of from 15 to 20 minutes' duration was performed on a cycle ergometer at high altitudes the maximum heart rate was usually reached in the half-minute after work began and was followed by a marked retardation and plateau. Paterson (1928), also using a cycle ergometer, observed in some cases an early maximum rate during work followed by a decline, after which a slow rise to the level of the early maximum took place. A pulse rate curve for light

work on the hand ergometer given by Gillespie, Gibson & Murray (1925) also showed this effect; and Backwell (1921 quoted by McDowall 1938) obtained similar results when the application of the effort was sudden. By the use of their cardiometer Boas & Goldschmidt (1932) found that in dancers the maximum rate occurred within one or two minutes of the commencement of the exercise, and it was only when the exercise was prolonged or became progressively more vigorous that the maximum rate appeared after a longer interval.

In none of the above instances can the postural effect have been responsible for the slowing during exercise, as no change in posture was involved. It is however possible that the passing off of the psychological tension which accompanies the commencement of effort may have been responsible, as presumably this component is also additive. This explanation would be especially likely in the experiments of Bowen, Backwell, and Boas & Goldschmidt, as either the nature of the exercise or the method of applying the load would tend to heighten psychological reactions.

The Postural Component.

This is itself due to two factors, the dynamic muscular movement of standing up and the steady gravitational factor when erect. The first would account for the primary

rise and fall of heart rate seen in Fig. 15, curve C; and the second would be responsible for the maintained increase in rate. It is, of course, the passing off of the dynamic factor which is responsible for the fall in heart rate during the brief standard exercise employed here.

In the present series the standing resting rate finally became steady at about 7 beats per minute above the sitting resting rate when counting was continued beyond the 25 seconds shown in curve C, Fig. 15. This is in general agreement with the average results given by previous workers for the sitting - standing increase. Vierordt (1906) quoted 9 beats per minute as an average figure, while Schneider (1916) gave 10 beats per minute for men, and Turner (1927a), 13 beats per minute for women. The whole question of postural increase in rate was exhaustively studied by MacWilliam (1933) who found wide variations in the sitting - standing increase, but 8 beats per minute was a usual figure for his subjects. His final conclusion was that "the vertical position of the thighs in standing is the essential condition of the quicker pulse rate." In his experiments the rate does not appear to have been counted in intervals smaller than half a minute however, and the brief initial rise and subsequent fall shown in Fig. 15 were thus not considered.

The Separation of the Postural and Exercise Components.

Helmreich (1923) seems to have been the first to recognise that even the simple act of standing erect involves two distinct components which may affect the heart rate. These caused " a dynamic rise corresponding to the increased use of oxygen seen in exercise, and a static rise corresponding to the changed relationship of the body to gravity". These components are clearly seen in the primary rise and subsequent plateau of the sitting - standing curve in Fig. 15. The existence of such a dual mechanism is also implicit in the results of Turner (1927b and 1929) though no quantitative data were given bearing on this particular point. In 1930 however, Turner, Newton & Haynes investigated the postural component alone by having the subject strapped to a tilting table so that no exercise was involved in the change of posture. They found that the delicacy of the circulatory reaction to gravity was such that a tilt of only 15 degrees produced a definite effect on the heart rate. This technique was extended to include exercise by Asmussen, Christensen & Nielsen (1939). Their subject was placed on a tilting bed and performed rhythmic arm movements against elastic resistance. The heart rate increased on tilting into the head-up position and if this tilt took place during the

arm work the postural increase was added to the exercise increase. The postural increase was abolished or reversed on clamping the legs. The authors ascribed the postural increase to diminished pressure in the central veins.

Thus, though a certain amount of work has been done on the separation of the exercise and postural components under artificial conditions, the present work would seem to be the first fully quantitative attempt to show that they have a distinct and additive effect on the heart rate during a natural mixed exercise.

SUMMARY.

The standard step-test includes two factors which may affect the heart rate;

- (a) The postural component, due to the change of posture from sitting to standing.
- (b) The exercise component, due to the actual stepping.

It is shown that each of these factors does influence the heart rate and that their effects can be separated but are additive in the mixed exercise. The fall in heart rate during the standard exercise is due to the partial regression of the postural component. The literature concerning these factors is discussed.

".....then the Pulse is disturbed, trembles or intermits, and seems to threaten every thing that is ill, whence one might believe that the Patient will be lost, when in a little time he perfectly recovers..... In Women with Child, and those who give Suck, there is a wonderful Variation of the Pulse, which may Seduce the Physician into capital Errors, if he forms a Judgment or Prognosis from the Pulse alone".

Boerhaave, (1756). p. 204.

CHAPTER 8.

SINUS ARRHYTHMIA AND SINUS BLOCK.

Even slight degrees of respiratory cardiac arrhythmia are very easily detected by the present method of recording. To determine its frequency of occurrence in Series A and B the tracings were divided into four categories according to whether sinus arrhythmia was absent, slight, marked or very marked.

The results were as follows:

Sinus Arrhythmia	Series A men (percentage)	Series B women (percentage)
Absent	9.3%	6.7%
Slight	29.3%	16.7%
Marked	54.7%	60.0%
Very Marked	6.7%	16.6%

Respiratory arrhythmia was thus present to a marked degree in over 60% of the men and in over 75% of the women. Fig. 17 shows a typical tracing. These percentages are higher than might have been expected from the literature. Thus Lewis (1925) states that in adult man the respiratory variation of pulse rate is inconspicuous or absent while the breathing is natural, but is universal during forced breathing. The question of forced breathing does not arise here as the exercise was so mild that it produced little effect on the respiration other

than a very slight and transient post-exercise increase in frequency and depth. The present results are more in agreement with Fleisch & Beckmann (1932) who found from accurate measurements of cardiac cycle length in man that respiratory arrhythmia was a constant physiological occurrence especially in subjects of vigorous physique. It would seem probable that the percentage of normal people showing sinus arrhythmia simply varies directly with the delicacy of the method of measurement employed. The arrhythmia invariably disappeared during the exercise (see Fig. 17) and this is in accordance with the majority of observations (Lewis 1925), though Schlomka & Reindell (1936) noted that sinus arrhythmia could persist in young people during mild and moderate exercise.

The general view seems to be that sinus arrhythmia is of no clinical significance and nearly always disappears on exercise (Roth, 1928; Treadgold, 1930). Mackenzie (1910) however, was of the opinion that well marked respiratory arrhythmia was a sign that the heart was free from disease. Tiitso & Pehap (1935) found that the strength of the arrhythmia at rest bore no relation to the work capacity of the individual. In view of these opinions it was considered worth while to compare the performances in the present test of ten subjects showing marked sinus

arrhythmia with ten showing no arrhythmia. The results are summarised in Table 3. The most obvious difference between the two groups is that the Early Resting Rate (2 min. before exercise began) and the Initial Rate are very much higher in those showing marked arrhythmia. This result was unexpected, as it is generally stated that the arrhythmia is most pronounced when the heart is beating slowly (Lewis, 1925; Boas & Goldschmidt, 1932; Fleisch, 1933; Cowan, 1939). That marked sinus arrhythmia occurs when the vagal tone is high was shown by Snyder (1915) and McDowall (1931), and that it is abolished by vagotomy was shown by Heymans & Samaan (1934). Earlier, Braun & Fuchs (1902) had observed that ordinary sinus arrhythmia was reduced or abolished after atropine. According to Katz (1941) the arrhythmia may persist even during rapid heart rates in youth and adolescence.

Although the Early Resting Rate and Initial Rate average about 10 beats per minute higher in those with marked arrhythmia, the rate 5 minutes after exercise shows a difference of less than 5 beats per minute. This suggests that those with marked arrhythmia have a greater psychological increase in heart rate in anticipation of the test, and this is borne out by the greater difference between the early resting rate and the immediate pre-exercise rate

in these subjects.

Since it is known that the respiratory variation, like the psychological increase in heart rate, is mediated mainly through the cardioinhibitory centre (see above) the fact that a marked sinus arrhythmia accompanies a marked psychological increase could be explained on the assumption that in such subjects the cardioinhibitory centre is unusually labile. That is, the centre might respond excessively to impulses from the higher centres (psychological increase), and from the respiratory centre (sinus arrhythmia). It was shown by Heymans (1929) that impulses irradiating directly from the respiratory to the cardiac centres do play an important part in the mechanism of sinus arrhythmia. In confirmation of this, Adrian & Buytendijk (1931) found that potential waves from the respiratory centre were propagated to a considerable distance. Other afferent impulses concerned in the respiratory arrhythmia come from the right auricle, (Bainbridge, 1920 and Starr & Friedland, 1946) and from the lungs themselves (Brodie & Russell, 1900). Excellent reviews of this field were given by Anrep (1936) and Anrep, Pascual & Rössler (1936).

As can be seen from the table, the average ages of the two groups were practically identical; there was thus no

evidence that the marked arrhythmia occurred in the younger subjects.

As regards the other indices, those showing marked arrhythmia have a higher maximum rate and post-exercise rate but the actual increase in heart rate due to the exercise is practically identical in the two groups. The acceleration during exercise is considerably greater in those with marked arrhythmia and this would tend to support the theory that these subjects have an unusually labile cardio-inhibitory centre.

Effect of Respiration on the size of the electrical variations.

It can be seen from Fig. 17 that respiration often causes a well-marked alteration in the size of the recorded R-S waves of the electrocardiogram. This effect was first pointed out by Einthoven, Fahr & de Waart (1913) and was confirmed by Waller (1913). They found that sometimes the R wave was larger on inspiration than on expiration and sometimes the reverse, depending to some extent on the leads employed. Rotation of the electrical axis of the heart during the respiratory movements was presumed to be the cause. With the chest leads used in the present experiments, the R wave usually decreases during inspiration but sometimes the reverse occurs.

Sinus Block.

Staehelin (1897), Cook & Pembrey (1913) and Dana (1919) described simple pauses in the rhythm of the heart, apparently dropped beats, which occurred in association with sinus arrhythmia or after exercise. Florence Buchanan (1909) observed similar diastolic pauses after training and in her view they largely accounted for the slow heart rate in trained subjects. About fifteen per cent of the subjects in the present series show this condition in some degree (Fig. 18) but not always in association with marked sinus arrhythmia. The condition is usually most prominent while the heart-rate is slowing down after exercise and may be similar to the step-like effect found by Mosler (1912) during the return to normal after more severe exercise. From the absence of extrasystoles or a compensatory pause the condition would appear to be some form of sinus block, perhaps due to sudden overactivity of the vagus as it resumes full control after exercise. Such hyperactivity might also account for the 'post-exercise trough' in the heart rate described by Klyver, Huang & Schafer (1927) and others.

SUMMARY.

Sinus arrhythmia is readily detected in the present tracings and was present to a marked degree in over 60 per cent. of the men and in over 75 per cent. of the women. The arrhythmia disappeared during exercise. The mechanism of sinus arrhythmia is discussed and it is shown that the phenomenon occurs in association with marked psychological variations in the heart rate. This suggests that it may occur in those with an unusually labile cardioinhibitory centre.

Simple pauses in the rhythm of the heart were present after exercise in 15 per cent. of the subjects. They were probably due to sinus block.

"Since pulses are readily altered in various ways - in fact, I might say there is no cause that does not change them - I have determined to take a threefold and very general difference in their change, and to speak specially about each in turn".

Galen, circa 170 A.D., in
"Libellus de Pulsibus ad Tirones".

CHAPTER 9.

THE EFFECTS ON THE HEART RATE OF VARYING THE LOAD,
SPEED AND DURATION OF THE EXERCISE.

The mean values and variability of the heart rate indices having been established by Series A and B, an investigation of the effects of load and of varying the speed and duration of the exercise was next undertaken.

Effect of Load. (Series W).

Method. Seventeen male medical students acted as subjects. Each first performed the standard exercise test exactly as in Series A and B (5 climbs at 96 steps per minute), carrying no load. A weight of 19.5 kg. was then placed in a haversack strapped to the subject's back and he rested quietly for 15 minutes during which it was found that the effects of the previous exercise on the heart rate had completely passed off. While the subject was seated the weight of the haversack was supported by a wooden block fixed to the back of the chair. At the end of the 15 minutes' rest the exercise was repeated carrying the weight of 19.5 kg. The same procedure was adopted with weights of 32.2 kg. and 40.3 kg. in the haversack. The average weight of the subjects was 64 kg. and the mean rates of working were:

With no load	-	478 kg.m. per minute.
With 19.5 kg.. Load	-	595 kg.m. per minute.
With 32.2 kg. Load	-	669 kg.m. per minute.
With 40.3 kg. Load	-	712 kg.m. per minute.

Results.

The results are given in Table 4 and graphs of the effect of increasing load on some of the indices are shown in Fig. 19. It will be seen from Table 4 that there is a slight increase in initial rate with each increment in load, due not to insufficient rest between the bouts of exercise but to psychological factors. This was shown by the fact that after each increase in load the resting rate rose a little higher as the time to commence work approached, although the subjects did not feel the load until they rose from the chair to begin exercise. It was therefore the anticipation of working with a greater load which caused the increase in initial rate.

There was a linear relationship between the maximum rate reached during the exercise and the load carried, whereas the mean acceleration of the heart rate remained relatively constant, Fig. 19. The post-exercise rate also increased linearly with load as might have been expected from its close correlation with the maximum rate. The increase of the remaining indices with load was quite definite but more irregular. Time to return to normal showed a particularly marked increase but this must be interpreted with caution as this index has already been shown to have a very marked intrinsic variability.

Graphs of the mean rates in 5 second intervals during the exercise with the various loads are given in Fig. 20. The curve for 'no load' shows the usual decrease in rate towards the end of exercise, caused by the withdrawal of the postural component. As the load increases this falling off in the steady acceleration diminishes until at 40.3 kg. it is scarcely perceptible. This is presumably due to the postural component being overwhelmed by the steadily increasing exercise component. In Fig. 19 it can be seen that as the load increases the average time of occurrence of the maximum rate moves towards the end of the exercise.

A point of interest which arises here is that the mean maximum rate with no load is 5 beats per minute less than in Series A, although the conditions in both sets of experiments were exactly the same. In the other special series also, when the standard exercise was employed, it was always found that the mean maximum rate was lower than in Series A. Several possibilities may account for this. The difference was usually about 5 to 8 beats per minute and this might easily have arisen by chance as it is approximately half the standard deviation. However, in order to make certain that the differences were not due to changes in the apparatus, the time-marker was re-calibrated

and found to be correct. Also, a considerable number of the Series A tracings were re-analysed in case improvement with practice in the technique of counting the beats had produced the difference. The original results were, however, confirmed and the difference must be accepted as a fact. One factor which may partly account for the lower rates in the subsequent series was that whereas the subjects in Series A and B were second-year medical students comparatively unused to human experiments, the subjects in the present series were third-year students taking the B.Sc. course and were therefore much more accustomed to such procedures. As it has previously been shown that psychological influences play a very large part in the control of the heart rate this may well have been a determining factor. For comparison with the results in Series A; the means, standard deviations and coefficients of variation of the indices during the basic standard exercise (5 climbs at 96 steps per minute with no load) in the load and duration series added together, a total of 30 male subjects, are given in Table 5.

Effect of Duration of Exercise (Series D).

Method. Thirteen male medical students acted as subjects. This series was intended to show the effect on the heart rate of varying the number of ascents of the steps, the rate of stepping remaining constant. No load was carried. Each subject performed three separate exercises with 15 minutes' rest between them. The first was the standard exercise of five ascents (exactly as in Series A), lasting approximately 20 seconds. The second consisted of 10 ascents lasting approximately 40 seconds, and in the third exercise the subjects made twenty ascents lasting about 80 seconds. In all cases the exercise began from the sitting posture and the rate of stepping was the standard one of 96 steps per minute.

Results. The results are given in Table 6 and graphs of the variation of certain indices with the number of climbs are shown in Fig. 21. Contrary to the results obtained in the load experiments, the mean initial rate here falls steadily from the first exercise to the last (Table 6). Apparently a simple increase in the duration of a light exercise with no load does not cause the psychological tension associated with load carrying. It will be seen from Fig. 21 that the mean values for maximum rate, percentage increase over initial rate and time to return to normal all increase with

the rise in the number of climbs. The rate of increase is not quite linear, tending to fall off as the number of climbs increases. The mean acceleration naturally falls as the exercise lengthens and the time to reach the maximum rate increases. All the other indices rise steadily with increase in the number of ascents.

The mean heart rates in five second intervals during the three exercises are graphed in Fig. 22. The curve for 5 climbs (the standard exercise) follows the usual course, whereas the heart rate continues to rise when the exercise is prolonged to 10 or 20 climbs. It will be seen that all three curves show the typical plateau due to the withdrawal of the postural component. This begins 12.5 seconds after the commencement of exercise and lasts for about 10 seconds when the exercise is prolonged beyond the usual 5 climbs, thus showing the same time relations as the postural increase curve (Fig. 15, curve C). This was of course to be expected, as the conditions during the first twenty seconds were exactly the same for all three exercises, and no swamping effect such as occurred in the load experiments was found here. It may also be noted that when 20 climbs were attempted very little change in heart rate occurred over the last twenty seconds of exercise, thus indicating that a "steady state" had been reached. During the return to normal after the exercises had ended there was very little difference in the slope of the

three curves, showing that though the mean time of return to the initial rate did increase with duration of exercise (see Fig. 21) the rate of deceleration remained constant during the first ten seconds after exercise ended. If these deceleration curves are extrapolated it will be found that they give values for the return to normal very close to those given in Table 6 which were obtained by the "micro-method" previously described. This indicates that the constant rate of deceleration was probably maintained until the initial rate was reached. Thus the difference in time of return to normal for the three exercises was simply an expression of the different levels to which the heart rate had risen at the end of exercise.

A general comparison of the results of the load and duration series would seem to indicate that an increase in the total work done brought about by load carrying has a greater effect on the heart rate than a similar increase due to extending the duration of the work. Thus in the load series, increasing the total work by 63% caused the following increases in the heart rate indices:-

Maximum Rate, 12.5 beats/minute; Absolute Increase, 9.0 beats;
Post-exercise Rate, 18.5 beats/minute; Return to Normal,
24 seconds.

In the duration series, increasing the total work by 100% increased the same indices thus:-

Maximum Rate, 6.4 beats/minute; Absolute Increase, 7.3 beats;

Post-exercise Rate, 6.6 beats/minute; Return to Normal, 7.5 seconds.

It must of course be noted that an increase in the load carried causes an increase in the rate of working whereas merely increasing the duration of exercise does not.

Combined effect of Speed and Duration of Exercise. (Series S).

Method. The other main factor which remained to be investigated was the effect of speed, in this case represented by the rate of stepping in the two-step test. A few trial experiments made it clear that if the rate of stepping was increased much over the standard 96 per minute, the subjects found great difficulty in keeping time with the metronome. On the other hand, if the rate was reduced to 30 steps per minute the movements became stiff and unnatural. Morehouse & Tuttle (1942) have recently found that in studies of post-exercise heart rate following various rates of stool stepping, rates of 40 - 50 steps per minute were required for acceptable reliability. With lower intensities of exercise the results were inconsistent. It was therefore decided to use only two rates of stepping, 48 per minute and 96 per minute and to make the subjects perform two exercises at each of these rates; one exercise of 5 ascents of the steps and the other of 10 ascents. Thus the effects of speed and duration of exercise could be compared on the same subjects. In order to avoid the complicating factor of change of posture at the beginning of effort, the exercises in this series began and ended in the standing position. No load was carried. Ten male medical students acted as subjects. They stood at ease for 5 minutes before each exercise began and again for 5 minutes after exercise ended. A break of

15 minutes was allowed between each of the four ascents, which were carried out in an irregular order to minimise any cumulative effects which might occur. In addition, each subject performed the standard exercise of 5 ascents at 96 steps per minute beginning and ending in the sitting posture for comparison with the results of the previous series. As the procedure was somewhat complicated it is summarised as follows:

Each subject performed:

- (a) 5 ascents of the two steps at 48 steps per minute,
standing to standing.
- (b) 10 ascents of the two steps at 48 steps per minute,
standing to standing.
- (c) 5 ascents of the two steps at 96 steps per minute,
standing to standing.
- (d) 10 ascents of the two steps at 96 steps per minute,
standing to standing.
- (e) 5 ascents of the two steps at 96 steps per minute,
sitting to sitting.

Results.

The average results for the various heart rate indices are given in Table 7, which also shows the mean total work performed and the mean rate of working. The variations in some indices with speed and duration of exercise are shown graphically in Fig. 23.

The initial rate in the exercises started from the standing posture averaged about 9 beats per minute higher than in those started from the sitting posture. This was in fair agreement with the mean sitting to standing increase of

7 beats per minute obtained in Series A (see Fig. 15).

When the total work remained constant at 182.3 kg.m. and the rate of working was approximately doubled, the mean maximum rate rose from 115.6 to 122.3, an increase of 6.7 beats per minute. When the total work was constant at 364.6 kg.m. and the rate of working was doubled the increase in mean maximum rate was 13 beats per minute, that is, very nearly twice the previous increase. (See Table 7).

On the other hand, when the rate of working remained constant at about 300 kg.m. per minute and the total amount of work was doubled the maximum rate rose by only 1.4 beats per minute, though when the higher rate of working of 590 kg.m. per minute was kept constant and the amount of work was doubled the rise in maximum rate was 7.7 beats per minute. Thus at the slow rate a two-fold increase in duration of work produced very little effect whereas at the fast rate it had a marked effect on the maximum rate attained. These results are also mirrored by the indices showing actual and percentage increase over the initial rate.

In the case of the return to normal a rather different result was obtained. When the total work was kept constant and the speed was doubled only small increases

in the return time occurred; whereas when the rate of working was kept constant and the total amount of work was doubled the increases were definitely greater. Thus when the total work was constant at 182.3 kg.m. and the rate was doubled the increase was only 2.1 seconds; but if the rate was constant at 300 kg.m. per minute and the total work was doubled the increase was 7.8 seconds. Similarly, when the total work was constant at 364.6 kg.m. and the rate was doubled the increase in return time was only 6.8 seconds; although when the rate remained constant at about 590 kg.m. per minute and the total work was doubled the increase was 12.5 seconds. As the return to normal is such a variable index these results must be accepted with caution but it will be shown in the discussion that they do agree with the results of some other workers in this field. The mean heart rates in 5 second intervals during the five exercises are graphed in Fig. 24. The effect of the postural component is again evident in the difference between the curves for 5 climbs at 96 steps per minute sitting to sitting and standing to standing. It is interesting that in the exercises of longer duration (10 climbs) the acceleration of the heart rate was definitely more sluggish than in the corresponding shorter exercises (5 climbs) at the same speeds, though the

conditions during the first 20 seconds were exactly the same. The subjects of course knew that the exercise was to be longer and these results might be due to a psychosomatic effect reducing the acceleration when the exercise was to continue for a longer time. Further, the times taken to perform the exercises were very nearly proportional to the number of climbs and the speed, so that the slower acceleration would not seem to have been due to the subjects taking the steps more slowly when they knew that the effort was to last longer. This effect is apparently only brought out in the standing to standing exercises when the complication of the postural component is absent, as it does not occur to any marked extent in the duration series , (Fig. 22) in which the exercise began and ended in the sitting posture.

DISCUSSION.

Many workers have attempted to correlate the work load with its effect on the heart rate. One of the earliest was Christ (1894), the exercise being a step test on special steps which displaced water so that the work done could be accurately measured, the pulse being taken before and after exercise by a sphygmograph. He concluded that with increased work the pulse rate increased up to a certain point, but this parallelism ceased at a certain figure of

work done when the pulse rate approached its maximum. Three years later much of this work was repeated by Staehelin (1897) whose findings agreed with those of Christ in so far as the general increase of heart rate with increased work was concerned, but Staehelin emphasised that there was no question of proportionality. He found great differences in the response in different individuals for a given amount of work, but the increases in heart rate with light, medium and heavy work were in that order. In 1911 Lowsley also found that the more vigorous the exercise the greater was the increase in pulse rate and the slower the return to normal. Similar qualitative results were obtained by Hedvall (1915), Brittingham & White (1922), Schneider & Clarke (1929), and Deppe & Bierhaus (1938). Tiitso & Pehap (1935) found that in trained subjects the steady heart rate reached during exercise did depend on the intensity of the work, but that in the untrained the heart rate increased continuously until the end of exercise. According to Schneider & Crampton (1936) pre-adolescent boys fail to show the linear relationship between load and pulse rate which is given by adults.

More definite quantitative evidence was obtained by Benedict & Cathcart (1913) who found that the pulse rate

even during severe muscular work closely followed the amount of the energy output measured in Calories per minute. Similarly Boothby (1915) obtained a practically linear relationship between the pulse rate and the intensity of work expressed as oxygen consumption per minute. Paterson (1928) and Bock, Vancaulaert, Dill, Folling & Hurxthal (1928) also obtained approximately linear correlations between heart frequency and metabolic rate. Recently Taylor (1941) obtained significantly high correlations between heart rate and work load for a range between 636 and 1191 kg.m. per minute and this was confirmed in 1946 by Erickson, Simonson, Taylor, Alexander & Keys, though in both cases the number of subjects was very small.

In practically all the references cited above the work was performed either on the bicycle ergometer or on some form of treadmill and the total amount of work was grossly in excess of that in the present series. Therefore, though the general conclusion that there is a more or less linear relationship between heart rate and work load is of interest, the findings are not strictly comparable with the present results. Comparison is however possible with the results of Peabody & Sturgis (1922) as regards the effect of load. Their exercise consisted of a climb of 60 steps

each 8 inches high in one minute, a step treadmill being employed. In eleven normal subjects (unloaded) the average rise in heart rate after 15 seconds of exercise was 16 beats per minute whereas when the subjects carried a knapsack weighing 23 kg. the corresponding increase was 21 beats per minute, a difference of 5 beats. In the present load series the difference between the increase with no load and with a load of 19.5 kg. is 5.3 beats, though the actual heart rates are considerably higher due to the higher rate of stepping (see Table 4).

None of the workers mentioned so far made any clear distinction between the effects of the constant work load or resistance, the duration of the work and the rate at which the work was carried out. The first to do this was Bowen (1903). He used two types of exercise, the bicycle ergometer and tapping a telegraph key, in both cases the resistance and the rate of working were varied independently. His general conclusion was that speed has a far greater influence on the pulse rate than resistance, and that this may be expected because for each muscle there is a certain resistance against which it works most economically (Heidenhain, 1892) and therefore as the resistance is increased up to that point the work may have proportionately less effect on the pulse rate. As regards

the effect of speed, he pointed out that as speed of movement increases the extra energy required to overcome inertia also increases rapidly and that this is bound to have an effect on the pulse rate. Cathcart, Richardson & Campbell (1924) found that when the amount of work done in unit time remained constant but the rate of performance varied, the mechanical efficiency was low with very fast and very slow rates and high with medium rates. Results similar to Bowen's were obtained by Cotton, Rapport & Lewis (1917a) who found that if the amount of work was kept constant and the rate of working was increased the maximum pulse rate varied accordingly. If, however, the amount of work was increased and the rate kept constant the changes lacked uniformity.

A very careful study of the whole question was published in 1925 by Gillespie, Gibson & Murray. Using a hand ergometer they investigated the effects of altering the load and the rate of work independently and also the effects of performing equal amounts of work with the load and rate varying. Readings of pulse rate (by palpation and auscultation) and blood pressure were taken before, during and after the exercise which lasted for 30 minutes. They found that the highest pulse rate attained increased with the load, but not strictly linearly in that the rate tended

to accelerate proportionately more with heavier loads, an effect which is not seen in the present load experiments possibly owing to the very great difference in the duration of the work. When the rate of working was varied they observed the maximum pulse rate to be directly proportional to it, as was the percentage increase in pulse rate. When the duration of work was varied (load and rate of work being constant) the pulse rate one minute after work ended was as a rule higher when the work was of longer duration, as in my experiments. They did not observe the time of return to normal but stated that the pulse rate fell more slowly after longer work-periods, in contrast to my results which show that the rate of deceleration is the same for all three durations of exercise.

Similar experiments on dogs under very accurately controlled conditions were carried out by Essex, Herrick, Baldes & Mann (1939) but their results were less consistent than those obtained by Gillespie et al. for man. A further differentiation between the effects of duration and speed of working was made by Merklen (1926). His conclusions were that: "...the intensity of the cardiac acceleration is governed less by the duration of the work carried out or its total amount, than by its momentary magnitude (*grandeur momentanée*) or by the rapidity of its execution, that is to

say by the power developed. On the other hand the duration of that cardiac acceleration or period of return to normal is much more under the influence of the duration than of the speed of working". The present results are in good agreement with this generalisation and in Fig. 25 it can be seen that the return to normal is influenced far more by the number of climbs (duration) than by the speed at which the exercise was performed.

As has already been pointed out, the severity of the exercise in practically all the above cases was far in excess of that performed by my subjects and it is thus interesting to note that the present results are in good general agreement with most of the previous findings. Moreover, the results are more consistent than in much of the earlier work. The clue to this may lie in an observation of Gillespie et al. (1925) that: "the differences in the rise of pulse rate with work varying in severity are more apparent in the rapid primary rise than in the subsequent slow increase which occurs as work proceeds". Thus detailed observation during comparatively short exercises may well give more consistent results than more widely spaced observations with heavier work, especially where maximum rate is concerned.

SUMMARY.

The effects on the heart rate of load carrying during the step test, and of varying the speed and duration of the exercise, were investigated. The variation of the different heart rate indices with load, speed and duration is discussed. In general, increasing the total work done by means of load carrying has a greater effect on the heart rate than a similar increase caused by extending the duration of the exercise. An increase in the speed of working, however, has a greater effect than either of the above factors. The time of return to normal is an exception, as it depends mainly on the duration of the exercise. These results, obtained with a brief, light exercise are in good agreement with those of other workers for more severe exertion.

"The skilful doctor knows what is wrong by observing alone, the middling doctor by listening, and the inferior doctor by feeling the pulse".

Sun-Szu-Mo, in the "Pulse Classic"
circa 2000 B.C.

CHAPTER 10.

THE EFFECT OF EXERCISE ON THE HEART RATE OF CARDIAC
PATIENTS WITH SPECIAL REFERENCE TO AURICULAR FIBRILLATION.

(Series C, B.C. and B.N.)

In order to compare the normal effects of exercise on the heart rate with those of patients suffering from various cardiac lesions, three sets of experiments were carried out. In the first set a number of ambulant cardiac patients performed the standard two-step test. These experiments were called Series C (ambulant cardiacs) and the results could be compared with those of Series A and B (normal men and women). The second series consisted of cardiac patients confined to bed, and a very light exercise (arm stretching) was devised for them. This was called Series B.C. (bed cardiacs). Finally, in order to give normal criteria for this bed cardiac series a group of healthy subjects performed the same arm stretching exercise in the recumbent posture. This was known as Series B.N. (bed normals).

In the cardiac patients the type of response to exercise varies so widely that it would be completely misleading to calculate a general mean for the heart rate indices. The cardiacs must therefore be divided into groups both by type of lesion (valvular disease, fibrillation,

heart block, etc.) and by clinical condition as regards amount of activity possible (good, fair, poor tolerance, etc.), before it is possible to take averages which have any meaning. Further, whether the patient is on digitalis or not must be considered, as this will alter the level of the heart rate throughout the test. Thus the cardiac patients must be dealt with in small groups.

One other point is that occasionally a comparison is made between the curves of heart rate in the cardiacs and in the normals (Series A and B). The average age of the normals is somewhat lower than that of the cardiacs, though the difference is not great as can be seen from the age columns in Tables 8 and 10. Moreover, Dill & Brouha (1937) and Robinson (1939) have shown that there is very little change in the heart rate response to moderate exercise over the age range covered by my subjects.

METHODS.

Series C. The subjects were forty ambulant patients with cardiac lesions of various types and grades. These patients performed the standard exercise of climbing two steps each ten inches high five times, the rate of stepping being 96 per minute. No load was carried, and the exercise began and ended in the sitting posture. The procedure was thus exactly as in Series A and B and the recording and analysis of the

tracings was carried out as described for these series (Chapters 3 and 4).

One of the objects of this investigation was to find out whether any of the heart rate indices or the curve of heart rate during exercise was correlated with the patient's clinical condition. Accordingly, an estimate of the patient's capacity for exertion was made and was called, for convenience, the 'Tolerance Estimate'. The following classification was adopted for all the Series C patients.

Excellent (E) - Symptomless, normal tolerance. No limitations.

Very Good (V.G.) - Symptomless, except on severe exertion.

Well compensated.

Good (G) - Symptoms only on moderately severe exertion.

Normally symptomless.

Fairly Good (F.G.) - Symptoms (especially dyspnoea) on long gradients. Not on level or normal stairs.

Fair (F) - Symptoms (especially dyspnoea) on stairs. Not on level unless walking fast.

Poor (P) - Easily tired and breathless on exertion.

Sometimes breathless even on level at normal walking speed.

These correspond roughly to Groups 1 and 2 of the Functional Capacity Classification of the American Heart Association (1926), but are somewhat more detailed.

Series B.C. This consisted of thirteen cardiac patients,

all of whom were confined to bed in hospital. The series was intended to provide a check on the results of Series C and to permit the investigation of the effects of treatment on the response to exercise under the controlled conditions of a hospital ward. The procedure adopted was as follows:-

The chest electrodes and stethograph were strapped on in the usual manner and the patient then lay resting quietly for fifteen minutes. Orthopnoeic patients were allowed to remain propped up at their accustomed angle but otherwise the subjects lay flat on their backs with the head supported by a small pillow and the arms by the sides. The exercise consisted in moving the arms from a position at shoulder level with the tips of the fingers touching the shoulders, to full stretch over the head at right angles to the plane of the bed. This arm stretching movement was repeated twenty times. The subject kept time to a metronome beating seconds so that each complete up and down movement occupied two seconds, and the whole exercise lasted forty seconds. The heart beats and respiration were recorded on a smoked drum in the usual way.

Series B.N. In this series twenty two medical students (ten women and twelve men) performed the arm stretching exercise described above. Each subject rested supine on the bed for fifteen minutes before the exercise was begun.

The heart rates in Series B.C. and B.N. were counted

in five second intervals before, during and after the arm stretching exercise, exactly as in Series A and B. The usual indices (initial rate, maximum rate, etc.) were also worked out for both series.

RESULTS and DISCUSSION.

Series B.N. For the reasons given above it will be necessary to divide the subjects of Series C and B.C. into groups according to the type of heart lesion and to discuss the results in each group separately. Series B.N., however, can be treated as a whole, and as it provides the normal for comparison with Series B.C. it will be dealt with first.

Fig. 25 shows the mean heart rate curve of the twenty two normal subjects in five second intervals before, during and after the 40 seconds of arm-stretching exercise. There is a sudden increase in rate at the beginning of exercise followed by a slight fall, probably due to the passing off of the initial psychological acceleration. The curve then climbs slowly to a maximum and after the end of exercise decelerates rapidly, the return to normal being accomplished in about twelve seconds. The general picture is as might be expected for normal subjects performing a very light exercise, and agrees well with the average type of curve obtained by Bowen (1903) for subjects tapping a telegraph key. It was found that this curve of heart rate was much more useful for comparison with the results of the bed cardiac

patients than were the mean individual heart rate indices such as maximum rate or actual increase in rate. The latter are therefore not tabulated here but are quoted when it is necessary to compare them with the results in the bed cardiac patients.

Series C and B.C. The results in the cardiac patients will be discussed under the following headings: (a) valvular disease of the heart; (b) angina pectoris; (c) coronary thrombosis; (d) effort syndrome; (e) heart block; (f) hyperthyroidism; and (g) auricular fibrillation. In each group the results will be given first for the ambulant patients (Series C) and then for the bed cardiacs (Series B.C.)

(a) VALVULAR DISEASE OF THE HEART.

Series C. This group consisted of twelve patients (7 males and 5 females) all suffering from simple valvular disease of the heart. Details of their condition will be found in Table 8. Their tolerance estimates varied from 'excellent' to 'fair', and none were on digitalis. Table 9 gives the mean values for the various heart rate indices in the four 'tolerance estimate' groups. There is obviously no inverse correlation between the clinical tolerance and the early resting rate or initial rate. In fact the poorer groups have consistently lower initial rates than the better groups. This result was unexpected and is difficult to explain, unless it be that the habitual limitation of exercise in the poorer groups allows the heart rate to settle at a lower level. It is also obvious that tachycardia is not inconsistent with excellent tolerance. Suarez, Fasciolo & Taquini (1946) found that only in cases of mitral disease did the basal heart rate vary directly with the degree of failure. As the 'very good', 'good' and 'fair' groups consisted largely of such cases, their results were not confirmed here. Allan (1925) showed by means of a model that tachycardia aggravates the effects of mitral lesions, but Albers (1942) found that the type of heart disease did not appear to have any essential influence

on the increase in pulse rate resulting from work. The mean initial rates are however higher than those of the normals in Series A and B (Early Resting Rate 83.0, Initial Rate 88.1, see footnote). This is in agreement with the results of Peabody & Sturgis (1922) and Woolham & Honeyburn (1927). It is noteworthy that the psychological increase in rate immediately prior to exercise which was such a conspicuous feature in the healthy subjects is almost completely absent in the V.D.H. cases. This is contrary to the findings of Peabody & Sturgis (1922) who noted no difference between the pre-exercise rise in cardiacs and normal subjects. It may be due to the fact that the initial rate is already so high. The variation in the mean maximum rate with the tolerance estimate is very small, though there is a distinct difference between the 'excellent' and 'fair' groups. Even more remarkable is the fact that the general level of maximum rate is, if anything, a trifle lower than in the normal subjects (131.4) but Kahn (1919) found that in compensated mitral regurgitation there might be only a very slight increase of heart rate after exercise. A somewhat similar result was obtained in two compensated

(Footnote: For comparison with Table 9, the corresponding average values for the normals (Series A + Series B) are given in brackets throughout this section in order to avoid frequent reference back to Table 1 where the full normal results will be found.)

cardiacs by McGuire, Shore, Hannenstein & Goldman (1939) and they suggested coronary insufficiency as a possible cause. The matter will be further discussed below in the section on the heart rate during exercise in anginal patients. The time to reach the maximum rate is, on the average, longer than in the normals (16.0); and the acceleration of the heart rate is slower (2.8). Neither index varies regularly with the exercise tolerance. The next three indices, extra beats produced by the exercise, percentage increase and actual increase on initial rate all rise as the tolerance estimate becomes poorer. The mean values in the V.D.H. cases are, however, considerably lower than in the normals (16.7, 51.2 and 43.4 respectively). This is probably due to the high initial rates of the cardiacs. Peabody & Sturgis (1922) using a slightly more severe exercise than the present one, found the mean actual increase in heart rate of patients with V.D.H. to be slightly greater than in normals but the differences were not marked. The post-exercise rate, which is higher than in the normals (95.0), increases steadily with decreasing tolerance. This is interesting as the maximum rate does not increase regularly, and presumably means that deceleration is delayed in the poorer tolerance groups. That this is the case is shown by the time to return to normal which increases

very markedly with decreasing tolerance (Table 9.)

In the 'excellent' and 'very good' groups the time of return is comparable with the normal average (23.4) but in the 'good' and 'fair' groups it increases very rapidly. This delayed recovery is in agreement with the results of Propst (1924), Woolham & Honeyburn (1927) and Bansi & Grosscurth (1930); though Spohr & Lampert (1930) found an increased recovery time in only 50 per cent. of their cardiac patients.

The effect of exercise on the cardiac output of cases with valvular disease has been studied by Means & Newburgh (1915), Grosscurth & Bansi (1932) and Lindhard (1937). The increase of stroke volume with exercise was generally less than in healthy subjects, but the mechanism by which it was brought about remained the normal one. Keys & Friedell (1939) employed the discrepancy between the stroke output of the heart and the net amount of blood circulated to measure the efficiency of the valves in such patients.

When the occurrence of sinus arrhythmia in the V.D.H. patients was examined, it was found that over all the incidence was much less than in the normals and that it diminished very markedly in the poorer tolerance groups as the following table shows:

Tolerance Estimate

Sinus Arrhythmia

	Excellent (2 cases)	Very Good (3 cases)	Good (2 cases)	Fair (5 cases)
Absent	0	0	0	4
Slight	1	1	1	0
Marked	0	2	1	1
Very marked	1	0	0	0

This would give some support to the theory (Mackenzie, 1910) that marked sinus arrhythmia is a sign of a healthy heart, though it must be emphasised that many normal subjects show no respiratory arrhythmia.

The Curve of Heart Rate during Exercise. (V.D.H. cases).

Graphs of the mean heart rates before, during and after the step test are given in Fig. 26. For the sake of clarity only three curves have been drawn; the 'very good' and 'good' tolerance groups whose graphs were very similar, being taken together and labelled 'good' for this purpose. For comparison, the heart rates in the normal subjects (Series A + Series B) are given in Fig. 27 drawn to the same scale. On the average, the initial acceleration in the cardiacs is slightly less than in the normals as was pointed out above. This is in agreement with the results of Borgard (1937) though the difference was far more marked in his cases. It will be noted from Fig. 26 that it is only in

the later part of the exercise that the tolerance groups become differentiated. There is an analogy here with the results of Taylor (1944). Working with normals of different fitness groups, he found that only during exercise did his heart rate curves sort themselves out to indicate the fitness groups correctly. The deceleration after the exercise shows considerable differences between the groups, as was brought out by the 'return to normal' index discussed above.

The Sitting - Standing Increase in the V.D.H. Patients.

For comparison with the normal response, the postural increase in heart rate between sitting and quiet standing was recorded in six of the V.D.H. patients. Two had 'excellent' tolerance and four had 'fair' tolerance. Fig. 28 shows the graphs of heart rate in 5 second periods for the change of posture in the six V.D.H. cases and in the seven normal subjects mentioned in Chapter 7. It can be seen that the actual increase on standing is much less and the acceleration slower in the cardiac patients but that the maximum rate reached when erect is much the same in both groups, though the sitting rate in the cardiacs is considerably higher. Cabot & Bruce (1907) found that the difference in pulse rate between the lying and standing postures largely disappeared in uncompensated valvular

disease and when the heart was seriously weakened from any cause. In the present instance the smaller postural increase in the cardiacs would seem to be largely due to the initial tachycardia. By subtracting the sitting-standing curves from the sitting-standing-exercise curves as in Chapter 7, the exercise components of the normals and cardiacs were obtained and are graphed in Fig. 29. The main difference between the two groups is that whereas the exercise component of the normals shows a steady acceleration up to the end of exercise, in the cardiacs there is a definite falling off in this component before the end of exercise. In Chapter 7 it was shown that the drop in rate during exercise in the normals was due to the falling off in the postural component, and it would therefore appear that there may be some other factor limiting the acceleration during exercise in the cardiacs. Separate graphs of the exercise component in the 'excellent' and 'fair' groups of V.D.H. patients are shown in Fig. 30. Both show the decrease in rate towards the end of exercise, and the higher maximum rate and slower deceleration in the poorer tolerance group are well shown.

Series B.C. V.D.H. Cases.

In the Bed Cardiac series there were four cases of simple valvular disease, three female and one male. None were on digitalis. Two were cases of mitral stenosis,

one had aortic incompetence and one mitral stenosis and aortic incompetence. All were confined to bed. Fig. 31 shows the average curves of heart rate during the 40 seconds of arm stretching exercise in the four V.D.H. cases (Curve A) and in the 22 normal subjects (Curve C).

It will be seen that there are considerable differences in the general shape of Curves A and C. In the patients with valvular disease (Curve A) the recumbent pre-exercise rate is extremely high, 114 beats per minute compared with 76 in the normals. During the exercise the patients' curve continues to rise rapidly for the first fifteen seconds and then flattens out. There is no evidence of the drop in rate soon after the beginning of exercise shown by the normals (Curve C). In the discussion on Series B.N. it was suggested that this fall was due to the passing off of the initial psychological acceleration. It was also shown above that very little psychological increase occurred in the ambulant V.D.H. patients, and the same was found to be the case here. In addition, any slight decrease in rate due to this cause would tend to be swamped by the greater exercise increase of the bed cardiacs.

The mean actual increase in rate due to the exercise was 25 beats per minute in the cardiacs and 14 in the normals, in marked contrast to the results obtained for

the ambulant V.D.H. patients. After the exercise the deceleration was much slower in the cardiacs, the return to normal taking 32 seconds as compared with 12 for the normals. It thus seems clear that even this very mild exercise imposed a much greater strain on the bed cardiacs.

The Effect of Rest and Treatment.

The effect of treatment on the response to the exercise was studied in two of the bed patients with valvular disease. The first patient was a woman of 33 with mitral stenosis and a dilated heart. Her curves of heart rate during the arm-stretching exercise are given in Fig. 32. The upper graph shows the response shortly after admission to hospital when the patient was confined to bed and was somewhat dyspnoeic. The lower curve was taken six weeks later just before dismissal. The cardiac condition was much improved and the patient had been allowed up for the past fortnight. The treatment given was simply rest and a light diet. It will be seen that the two curves are very similar, even to a tendency for the rate to become fixed over several intervals. The later curve is largely a repetition of the earlier, but at a much lower general level of heart rate. The clinical improvement here has therefore had very little effect on the acceleration of the heart during the exercise but has enabled that acceleration to take place from a much lower baseline than before. The return to normal is, however,

more complete in the later tracing.

The second patient was a woman aged 25 suffering from mitral stenosis and aortic incompetence. Graphs of her heart rate during the exercise are given in Fig. 33. The upper curve was recorded six days after admission when the patient was orthopnoeic and the liver was large and pulsatile. The exercise caused little acceleration over the very high initial rate and the deceleration after exercise was very slow. The lower curve was taken two weeks later after treatment with Guy's pill. The day before this tracing was recorded the pulse showed some irregularity and two days later definite coupling appeared. The initial rate is very much reduced, probably mainly due to the digitalis, but the response to exercise is now very much greater and the deceleration after exercise is considerably more rapid. In this case, therefore, there was a definite change in the pattern of acceleration during exercise, as well as a lowering of the baseline. It is also interesting that though in this case digitalis was pushed almost to the point of coupling at the time of recording, the heart rate was still very mobile.

The heart rate curve from an additional bed cardiac with valvular disease is given in Fig. 31, Curve B. The patient was a man aged 22 suffering from aortic incompetence. At the time this tracing was taken he was

convalescent and was allowed up for four hours per day. His curve closely resembles the normal bed exercise graph (Curve C), but the actual increase in rate throughout the exercise is somewhat greater.

In general, it would appear that as treatment succeeds and the cases become convalescent the curve of heart rate falls towards the normal. Karpovich, Starr & Weiss (1944) and Karpovich, Starr, Kimbro, Stoll & Weiss (1946) found that the post exercise rate after a step test was a useful guide to convalescence after rheumatic fever, contrary to the earlier results of Mann (1918).

(b) ANGINA PECTORIS.

Series C. Graphs of the heart rate during the standard two-step test in the three cases of angina pectoris are given in Fig. 34, together with the mean curve for normal males. Curve A was from a male aged 42 whose clinical tolerance was estimated as 'fair'. The electrocardiogram showed numerous ventricular extrasystoles but was otherwise normal. Curve B was from a man aged 56 whose tolerance estimate was 'poor'. There was marked arteriosclerosis and X-ray showed an enlarged heart and uncoiled aorta. The electrocardiogram showed left ventricular preponderance. Curve C gives the response in a man aged 31 with a 'fairly good' tolerance estimate. A double aortic murmur was present, probably due to syphilitic aortitis. The electrocardiogram showed marked left ventricular preponderance and the P-R interval was normal.

The main feature of all three curves from the anginal patients is the low maximum rate during the exercise, the mean being about 15 beats per minute lower than in the normals or the simple V.D.H. cases. The rate of deceleration was about the same as in the V.D.H. cases.

Eppinger, Kisch & Schwartz (1927) pointed out that certain anginal patients are characterised by inability to increase the heart rate normally during exercise, and

Proger, Minnich & Magendantz (1934) confirmed this and also found that atropine increased the heart rate but not the capacity for work in such cases. They also noted the development of extrasystoles during exercise in some cases shortly before the onset of pain and their disappearance before the end of exercise. Ventricular extrasystoles were seen in two of my cases but they disappeared during the exercise as this irregularity usually does. In none of my cases did the standard exercise produce any anginal symptoms, however. In order to test this finding of Proger et al. one of my anginal patients performed rapid stepping until the onset of typical pain, but there was still no evidence of extrasystoles during the exercise, though a burst of them did occur about three minutes after the end of exercise. Such extrasystoles appeared to be associated with emotion rather than with the exercise itself. Fig. 35 shows a typical burst of extrasystoles which occurred when one of the angina patients was suddenly spoken to. An electrocardiogram from this subject (lead LV F) showing that the extrasystoles arose from both ventricles is also included in the figure.

Bierring, Larsen & Nielsen (1936) investigated the changes in heart rate after maximal exercise on the bicycle ergometer in two cases of angina. The main feature was an unusually rapid drop in heart rate to below the resting level,

accompanied by transient gross changes in the E.G.C. This effect was not seen in my cases in which the mean time of return to normal was 39.5 seconds compared with 21.4 seconds in the normals. Exercise has been used by Holzm~~ann~~ & Wahrmann (1936), Schott (1939) and Evans & Bourne (1941) to accentuate abnormalities in the electrocardiogram. They found that a fairly strenuous exercise test produced changes in the E.C.G. suggesting myocardial disease in about half the cases showing no abnormality at rest. Riseman & Brown (1939) showed that the breathing of oxygen increased the capacity for work of patients with angina pectoris. Bourne & Scott-Bodley (1938) have pointed out that angina of effort is in itself not a dangerous symptom and that the prognosis depends entirely on the underlying vascular disease.

With regard to the limitation of heart rate during exercise in the patients with angina the most obvious explanation would be that the inadequacy of the coronary circulation in some way limits the acceleration of the heart as suggested by McGuire et al. (1939) for those cases of simple valvular disease in which the heart failed to accelerate normally. Anrep & Segall (1926b) showed that in the innervated heart-lung preparation acceleration of the heart did not augment the coronary flow. Landis, Brown,

Fauteux & Wise (1946) found that after ligation of coronary arteries in the dog the resting heart rate rose, but the increase of heart rate due to a standard exercise became less. According to Wayne & Laplace (1933) the appearance and disappearance of anginal pain is much more closely related to the heart rate than to the arterial pressure. Thus the limitation of the rate during exercise might play a definite part in delaying the onset of pain. Against this theory is the fact that in coronary thrombosis the heart rate may be either normal, rapid or slow. If it is slow the cause is usually the development of heart block (White, 1937). Careful examination of the present tracings during the exercise period showed no evidence that the limitation of rate was due to dropped beats, as would be expected if a partial heart block had occurred. A point from the present series in favour of the coronary insufficiency theory is that in two of the three cases there was definite evidence, X-ray or electrocardiographic, of left ventricular hypertrophy. Harrison, Ashman & Larson (1932) showed that in hypertrophy there is probably a relative inadequacy of blood supply to the heart muscle even in the absence of narrowing of the coronary arteries. In addition, one of my cases had aortic regurgitation and as Fishberg (1937) pointed out, the low diastolic pressure in

the aorta in this lesion may well cause inadequate coronary flow. It is interesting that the cases with hypertrophy (Curves B and C in Fig. 34) have the lowest maximum rates during the exercise and the most rapid deceleration after it, while the case of aortic regurgitation (Curve C) has the lowest maximum of the three. While this is perhaps worth pointing out, it is appreciated that these differences are not great enough to be significant in such a small series.

The case of aortic regurgitation (Curve C) shows a low maximum rate during the exercise, and a rapid drop in rate after the exercise, as might have been expected from the low heart rate during the standard exercise (Fig. 34, Curve C). It shows the same low maximum rate as the other patients, but also a rapid drop in rate after the exercise to a level far below the initial rate, as described by Loring et al. (1936) for their angina cases.

(c) CORONARY THROMBOSIS.

Series C. It was only possible to obtain a record from one case of coronary thrombosis. The patient was a man aged 28 who had been twice in hospital, the first time with undoubted coronary thrombosis confirmed electrocardiographically. Two years later he was again admitted with a complaint of dyspnoea and cardiac pain. The E.C.G. showed multiple ventricular extrasystoles and slurring of the QRS complex. The tolerance estimate was 'poor' when the present record was obtained five months after his second admission to hospital. A portion of his tracing is given in Fig. 36, and shows that multiple ventricular extrasystoles were still present with a definite tendency to coupling of beats. The extrasystoles disappeared for short periods during and immediately after the exercise, as might have been expected. The curve of heart rate during the standard exercise is given in Fig. 37, Curve C. It shows the same low maximum rate as the angina patients, but also a rapid drop in rate after the exercise to a level far below the initial rate, as described by Bierring et al. (1936) for their angina cases.

(d) EFFORT SYNDROME.

Series C. There were two cases of effort syndrome. Both were men, one aged 41 and the other 44. They complained of chest pain and dyspnoea on exertion and both had a history of nervous breakdown. Their tolerance estimates were 'good' and 'fairly good' respectively. The curves of heart rate during the step-test are shown in Fig. 37, Curves A and B,

Opinion seems to be sharply divided concerning the effect of exercise on the heart rate in effort syndrome. Thus Meakins & Gunson (1917), Cotton, Rapport & Lewis (1917a), Parkinson (1917a), Lewis (1917) and Bourne (1940) found that their patients had high resting rates and a greater rise of rate after exercise with a slower return than had normal subjects. On the other hand Mabon (1919), Parkinson (1941) and Wood (1941) found little or no abnormality in the post-exercise rate or the return to normal, though their subjects also had high resting rates. Parkinson (1917b) showed that the tachycardia was not controlled by full doses of digitalis. In the present instance both subjects had initial rates of over 100 per minute, but the maximum rates recorded during exercise were not abnormally high. The deceleration after exercise was delayed in both cases and the times of return to normal were 49.5 seconds for Curve A and 27 seconds for Curve B (Fig. 37). Thus the one feature of this condition on which

all observers agree is the high resting heart rate. This would favour the psychological explanation of effort syndrome as put forward by Wittkower, Rodger & Wilson (1941), Chamberlain (1941) and Jones & Scarisbrick (1942); though their views were not entirely accepted by Cohn et al. (1944). As was brought out in the Royal Society of Medicine discussion on effort syndrome (1941), the effects would appear to be due to central stimulation rather than to hypersensitivity of any peripheral mechanism.

(e) HEART BLOCK.

One case of complete heart block and two cases of partial heart block were investigated.

Series C. The patient with complete heart block was a man aged 56 whose tolerance was estimated as 'fairly good'. This subject performed the standard step test on two separate occasions at an interval of almost exactly one year. On the second occasion an E.C.G. taken at rest gave an auricular rate of 69 beats per minute and a ventricular rate of 32 beats per minute. The heart rates during the step test are shown in Fig. 38, Curves C and D. It will be seen that the heart rates at rest are identical on both occasions and are absolutely constant, but that the exercise does succeed in increasing the rate by a very small amount. On the second occasion the patient stated that he felt definitely better than during the previous year and this is possibly correlated with the slightly greater flexibility of the ventricular rate (Curve C, Fig. 37). Gilchrist (1934) found that in patients with complete heart block the greater the increase in ventricular rate on exercise the less incapacitating were the cardiac symptoms. In the present case, however, the actual difference in maximum rate on the two occasions was too slight to permit of any definite conclusions being drawn.

During the first examination of this patient, in addition to performing the standard 5 ascents of the two steps

he also performed a second test consisting of 20 ascents at the standard speed. This quadrupling of the usual exercise caused marked dyspnoea and was undertaken to see whether more severe exercise would force the heart rate still higher. Graphs of the heart rate during the standard exercise (5 ascents) and the prolonged exercise (20 ascents), performed within half an hour of each other, are given in Fig. 39. Curve B shows the slight but definite increase in rate caused by the standard 5 ascents. No extrasystoles were seen throughout this tracing. Curve A shows the effect of 20 ascents (exercise to breathlessness). The rise of heart rate here is much greater and tends to occur in a series of steps, but there is a good deal of minor variation in rate during the exercise. When exercise ends the rate returns fairly smoothly to a baseline about 3.5 beats per minute above the initial rate. Then, beginning about 45 seconds after the end of exercise the smooth curve is interrupted by sudden increases in rate reaching a maximum of 50 beats per minute. These occurred, of course, while the patient was still sitting quietly and were due to extrasystoles giving the appearance of occasional coupled beats. This portion of the tracing is reproduced in Fig. 40. Typical ventricular extrasystoles are seen, and the length of the returning cycle is exactly equal to that of the

ideoventricular rhythm itself, the usual finding in complete heart block (Lewis 1925). Such extrasystoles did not appear either before or during the prolonged exercise itself, and none were seen on the tracings before, during or after the standard (5 ascents) exercise in this patient.

The second case was one of partial heart block in a woman aged 66 suffering from dizziness and hypertension (S.B.P. was over 200 m.m.). Her clinical tolerance estimate was 'poor'. The heart rate during the standard step test is shown in Fig. 38 Curve B. As might have been expected the resting rate before exercise is more variable than in the patient with complete block, but the true acceleration produced by the exercise is very little greater than in the previous case. The sudden acceleration at the end of exercise is entirely due to a single ventricular extrasystole. No other extrasystole occurred throughout the tracing.

Series B.C. The third case was a man aged 74 who was admitted to hospital after collapsing in the street. The diagnosis was coronary artery thrombosis, and E.C.G. indicated an infarction on the posterior aspect of the heart. The pulse rate on admission was only 40 per minute, but it increased gradually until when the tracing was taken on the fourth day it was just over 50. The infarction would appear to have caused a varying degree of block as the heart rate

was sometimes irregular and the single E.C.G. which was taken two days before the tracing showed no definite evidence of block. However, it would seem that at the time the present tracing was recorded a high degree of block was present as is shown by the absolute steadiness of the resting rate (Fig. 38, Curve A). The standard arm-stretching exercise produced a variation in the rate, which after swinging up and down during the first part of the exercise finally settled at 54 beats per minute. Almost immediately after the end of exercise the rate dropped to the steady pre-exercise value of 51.6 beats per minute. No extrasystoles were seen throughout this tracing.

The main conclusions with regard to the heart block patients were that a very mild exercise did produce some acceleration in all the cases, that the onset of this acceleration was delayed compared with the normal, and that the pre-exercise emotional rise of heart rate did not occur.

DISCUSSION:

The fact that the cardiac nerves can influence the ventricular rate in complete heart block was demonstrated by Hering (1905 a and b). He found that in the dog stimulation of the accelerator nerves had the more definite effect. In the following year Wihl (1906), also working on dogs, showed that vagus stimulation slowed the ventricles in complete heart block. Einthoven & Wieringa (1913) found

that vagal stimulation could affect not only the main bundle but also its branches. In the turtle heart Izquierdo (1929) found that strong sympathetic stimulation gradually removed the heart block after a long latent period. That the more natural stimulation of exercise could also increase the ventricular rate in dogs in which the bundle of His has been cut was shown by Erlanger & Blackman (1910). In spite of this, many of the standard textbooks on heart disease continued to doubt whether exercise could increase the pulse rate in complete heart block in man. Frédéricq (1912) however, showed that in man such an increase could occur, and this was confirmed by Liljestrand & Zander (1927). The latter described the increase of heart rate on exercise in an athletic young man with complete heart block confirmed electrocardiographically. The ventricular rate rose rapidly to double the resting value and the electrocardiogram showed that the increased rate was caused by acceleration of the ideoventricular centre and not by extrasystoles. The authors realised that this case was unusual in the rapidity and degree of acceleration produced and suggested that the lesion must have been in the A.-V. node itself, leaving the distal portion of the node both in connexion with the cardiac nerves and with the Bundle of His. Gilchrist (1934) reviewed the literature on heart-block and exercise and added several cases of his own. He used a step test somewhat similar to the present one and

found that exercise did increase the ventricular rate in complete block but that the degree of response varied considerably in different individuals. He did not, however, record the heart rate during the actual exercise.

As it is unusual for any great increase in heart rate to take place in such patients the question of their cardiac output during exercise is of interest. In Liljestrand & Zander's patient the cardiac output responded normally but, as was pointed out above, this was clearly an unusual case. Alt, Walker & Smith (1930) determined the cardiac output in two cases of complete block during exercise on the bicycle ergometer. In one case the ventricular rate doubled during exercise, due partly to the occurrence of extrasystoles, and the output per beat was only moderately increased. In the other case the heart rate fell slightly during exercise and this was accompanied by an enormous increase in the stroke volume, the value reached being the highest reported in the literature. Confirmation was thus obtained for the theory, long held, that failure to increase the heart rate must be compensated by increase in stroke volume. This would account for Herxheimer's observation (1932) that in a few instances outstanding athletic performances have been given by subjects with total heart block.

With regard to the effect of the cardiac nerves on heart block in the human subject there seems little doubt that

the vagus can exert a definite influence in cases of partial block, the P-R interval being shortened by exercise (Parkinson & Drury, 1917) or the conduction defect may even be abolished (Allan, 1928). According to Miller (1942b), Logue & Hansen (1944) and Halminen (1945) atropine also shortens the P-R interval in many cases of partial or intermittent block, but fails to have any effect in cases of chronic complete heart block. The marked individual variation in the heart rate response of such patients can probably be explained by the varying distance of the lesion from the A-V node. The nearer the lesion is to the node the greater would be the response, as is well illustrated by the case of Liljestrang & Zander quoted above.

Certain of the features of the cardiac acceleration shown by my heart block patients during exercise could be explained on the theory that in this condition the sympathetic innervation plays a more definite role than usual. This would explain the gradual and delayed onset of the acceleration, as it has been shown by Hunt (1899) and Samaan (1935b) that this is a characteristic of sympathetic stimulation of the heart. It would also account for the absence of the psychological acceleration which is primarily due to the withdrawal of vagus restraint (Bowen, 1903, Samaan 1935a). In two of the present tracings the heart rate was augmented by extrasystoles, in one case during the

exercise and in the other a considerable time after exercise had ended. Such extrasystoles in cases of heart block have been noted by Wenckebach & Winterberg (1927), and they occurred during exercise in one of the two cases investigated by Alt, Walker & Smith (1930). This phenomenon could also be explained by the action of the sympathetic, as it is known (Howell 1946) that stimulation of the sympathetic may produce extrasystoles by enhancing the activity of ectopic pacemakers. As a result of one experiment, Frédéricq (1912) concluded that exercise acceleration of the heart rate in a dog with experimental heart block was due solely to the accelerator nerves, but his evidence was by no means conclusive.

(f) HYPERTHYROIDISM.

Series B.C. The effect of treatment on the response of the heart rate to bed exercise was studied in two patients with Graves' disease.

The first case was a woman aged 22 who had a B.M.R. of +43%, much tremor but no exophthalmos. The blood pressure was 125/68. The curves of heart rate during the standard arm-stretching exercise are given in Fig. 41. Curve A shows the response in the untreated patient. The initial and post exercise rates are very high but the acceleration during exercise is slow and the actual increase in rate is only about half that seen in the control series. Curve B was obtained three weeks later after the patient had been treated for five days with Lugol's iodine (five minims t.i.d.). The B.M.R. was now +30% and tremor was still present. The blood pressure was unchanged. The graph shows that the initial and post exercise rates have greatly diminished, but the most striking feature is the flatness of the curve during exercise. The actual maximal increase in rate remains the same as in Curve A but is mainly due to a sudden transitory increase late in the exercise. It will be noted that this increase passes off while the exercise is still in progress. Curve C was taken 12 days after Curve B and eight days after the operation of partial thyroidectomy. It will be seen that the graph is now very similar to that of

the normal subject performing the bed exercise.

(c.f. Fig. 25). The acceleration at the beginning of exercise is now rapid and the actual increase in rate is much greater than in the two previous curves.

The second case of hyperthyroidism was a man aged 46. He was nervous and excitable, had lost weight, and complained of palpitation and breathlessness on exertion. There was slight exophthalmos. The B.M.R. was +14%. In view of the somewhat atypical heart rate findings it should be pointed out that a partial thyroidectomy produced marked improvement and the clinical diagnosis was not in doubt. The first bed exercise test was performed shortly after admission and before any medication had been given (Fig. 42, Curve A). The resting heart rate was below 70 per minute in spite of the other symptoms of hyperthyroidism, and this was confirmed over several days. It is possible that this case belonged to the 'vago-tonic' type of Graves' disease mentioned by Fishberg (1937), in which the cause of the slow pulse is obscure. The entire curve for the exercise is very similar to the normal one as regards shape and maximum rate reached, though the actual increase in rate is greater because of the low initial rate. The second bed exercise (Fig. 42, Curve B) was performed ten days after the operation of partial thyroidectomy. In this case the initial rate is much higher and the acceleration due to the exercise

is very slight. The increased resting rate was accounted for by the fact that this patient had a febrile attack shortly after the operation and the pulse rate was still affected by this when the test was performed. It is interesting, however, that under these circumstances the exercise produced only a very slight and transient increase in heart rate.

DISCUSSION.

According to Fishberg (1937) it is generally agreed that the tachycardia of hyperthyroidism is part of the regulating mechanism by which the cardiac output is raised to the level required by the increased metabolic rate. An editorial in the British Medical Journal (1942) summarised the evidence that the raised cardiac output in Graves' disease is entirely maintained by the tachycardia, the stroke volume remaining unchanged. It was shown by Yater (1931) that thyroxin has a specific effect on the heart muscle fibres. He found that if the heart of a hyperthyroid rabbit was isolated and perfused it beat much faster than the isolated heart of a normal rabbit, and that the accelerated rate persisted even after cutting the Bundle of His. This work was later confirmed by Leblond & Hoff (1944). In the present cases perhaps the most striking feature is the manner in which the heart rate mirrors the changes in metabolic rate, though this is more evident in the resting heart rates than in the actual response to exercise. It is of interest that

both the hyperthyroid patients have one test in which the resting heart rate was raised very little by the exercise. In the first case (Fig. 41, Curve B) the patient was under treatment with iodine. According to Means & Lerman (1938) and Goodman & Gilman (1941) the action of iodine in thyrotoxicosis is to inhibit the passage of thyroxine from the gland into the blood. The patient's condition under iodine treatment would then closely resemble that after partial thyroidectomy. But in this patient after operation the heart rate became mobile again (Fig. 41, Curve C). In the second case (Fig. 42, Curve B), the failure of the heart rate to respond to exercise occurred after thyroidectomy. The factor which was common to both cases was a high resting rate. It is therefore probable that the failure to respond was due to the heart rate being already sufficient to carry the circulatory needs of the exercise when the disturbing influence of the hyperthyroidism was removed and in the absence of organic heart disease.

(g) AURICULAR FIBRILLATION.

The response of the heart rate to exercise in patients with auricular fibrillation is of great interest because of the abnormal mechanisms involved. In Series C (standard step test) twenty tracings were recorded from thirteen ambulant patients with fibrillation. In Series B.C. (bed cardiacs, arm stretching exercise) only three fibrillation patients could be obtained, but in one of these eight serial tracings were taken showing the effect of treatment over a period of more than a month. Table 10 gives details of all these patients including their estimated tolerance groups.

RESULTS.

Series C. (Ambulant patients).

Table 11 gives the usual heart rate indices in the fibrillation cases grouped by tolerance estimate for comparison with similar data given in Table 9 for the cases of valvular disease and in Table 1 for the normal subjects. Unlike the V.D.H. cases, many of the fibrillation patients in the poorer tolerance groups were under treatment with digitalis and these are tabulated in separate columns in Table 11. There is no definite correlation between any of the indices and the tolerance estimate in the fibrillation patients. This was the expected result, as the completely

irregular ventricular rhythm characteristic of fibrillation produces sudden arbitrary changes in heart rate in the successive five second intervals counted. Certain general characteristics, however, can be seen in Table 11. Apart from the single case with 'good' tolerance the maximum rates reached during the step test were much higher than in either the normals or the V.D.H. cases. This also holds good for the percentage and actual increases over the initial rates, and for the post-exercise rates. The time to reach the maximum rate is generally prolonged compared with the normal series and the acceleration of the heart rate, though slightly less on the average than in the normals, is definitely greater than in the V.D.H. cases. Owing to the complete irregularity of the heart rate the method of calculating the time of return to normal is valueless in fibrillation and so this index is not included here.

The Curve of Heart Rate during Exercise.

Fig. 43 shows the mean curves of heart rate during exercise for the different tolerance groups in all the fibrillation patients who were on digitalis. For comparison, the mean curve of the normal subjects is also included in the figure. Two of the curves ('Fairly Good' and 'Fair') show a distinct fall in heart rate during the first five seconds of the exercise, a phenomenon which is of course never seen

in normal cases. In general, compared with the normal curve, the acceleration immediately after the beginning of exercise in the digitalized fibrillation patients is less; but later in the exercise very marked acceleration occurs, reaching a higher value than in the normals. Thus in Fig. 43 all the curves except 'poor' show a steeper slope in some portion of their course than does the normal curve. This sudden delayed acceleration was very characteristic of the fibrillation cases and will be discussed in detail later. It will also be seen that after the end of exercise the heart rate falls very slowly compared with the normal, especially in the poorer tolerance groups. There was no correlation between the estimated tolerance and the behaviour of the heart rate during exercise as shown by these curves. As was pointed out above, this was to be expected; and in any case different degrees of digitalization in these patients might well mask any such correlation.

The curves of heart rate during the step test in four fibrillation patients who were not on digitalis are given in Fig. 44. In general they show the same characteristics as the digitalized patients; these include the drop in rate at the beginning of exercise (in two cases), the marked delayed acceleration (in three cases), and the continuation of a high heart rate after the exercise is over.

Curve A is an exception as regards acceleration but this may be linked with the fact that it starts from the highest initial rate of all. Curve B, from a patient with mitral stenosis and fair tolerance has the highest maximum rate reached by any patient, 235.2 beats per minute averaged over a five second period. A portion of the actual tracing is given in Fig. 45. It is surprising that such an extreme frequency was reached during a very mild exercise lasting only twenty seconds in a patient who could get about reasonably well.

In both the digitalized and non-digitalized groups the curves of heart rate for the patients with 'poor' tolerance do not as a rule show such high instantaneous values of acceleration as the other tolerance groups.

Series B.C. (Bed cardiacs).

Fig. 46 shows the curves of heart rate during the standard arm-stretching exercise in two cases of auricular fibrillation, one on digitalis (Curve B) and one not (Curve A). A fall in rate at the beginning of the exercise occurs in one case and both show the typical delayed steep acceleration. In general, this very light exercise produced the same kind of curve in the bed patients as did the step test in the ambulant patients. This is well illustrated in Fig. 47. Curve A shows the response of a fibrillation patient to the step test and Curve B the response of the same

patient to the bed exercise test eighteen months later after he had been admitted to hospital. The general similarity of the two curves is evident. The normal curves for both step and arm-stretching exercises are also included in the figure (Curves C and D), and the delayed exaggerated response of the patient to both exercises as compared with the normal can be seen.

Variations in a Single Subject.

Series C. It might be expected that owing to the complete irregularity of the ventricular rate in fibrillation there would be very great variations in the shape of the heart rate curve even in the same patient on the same day. Fig. 48 shows three curves of the response of the heart rate to the standard step test taken at fifteen minute intervals in the same fibrillation patient. The curves were taken in the order A, B, C. The curves do indeed show some variation, but not perhaps so much as the complete arrhythmia would lead one to expect. In general, the acceleration tends to decrease in successive tests. These results were confirmed in two other fibrillation patients.

The Effect of Clinical Condition.

Series C. It was shown above that there was no correlation between the heart rate curves and the clinical tolerance of different fibrillation patients. In view of the fairly consistent curves given by any individual patient however, it

seemed possible that clinical improvement or deterioration in a given subject might be reflected in his heart rate response. It was possible to try this in two of the ambulant fibrillation cases. The heart rates during the standard step test are shown in Fig. 49. Curve A is from a patient on digitalis whose tolerance classification was 'fairly good'. Curve B is from the same patient one year later. He was still receiving the same dose of digitalis and had felt gradual improvement in his condition since Curve A was taken. This improvement may be reflected in the generally lower level of Curve B. Curve C shows the response in another fibrillation patient also on digitalis. His tolerance classification was 'poor'. Marked coupling of beats was present throughout this tracing. Curve D is from the same patient six months later when his general condition had become definitely worse. He was still on the same dosage of digitalis. In this case the second tracing shows a much greater acceleration of the heart rate by the exercise, and no coupling was seen. Though these results do suggest that the heart rate curves of these patients may vary with their clinical condition it is also possible that the variations were due to different degrees of digitalization, though the actual dosage did not vary. This explanation would be especially likely in the second case. An interesting feature of this figure is the way in which the

characteristic shape of the curve for each patient is maintained even after a long interval and in spite of changes in clinical condition. Thus curves A and B both show a 'peak' type of curve with marked oscillations after the end of exercise, whereas Curves C and D are of the 'plateau' type with a relatively smooth post exercise fall in rate. The tracing from which Curve C was taken showed the actual onset of a period of coupling. A reproduction of part of this tracing is given in Fig. 50 and the commencement of coupling in a burst of extrasystoles can be seen to occur about 35 seconds before the beginning of exercise. The coupled beats persisted through^{out}/exercise and were still present when the patient left the laboratory some fifteen minutes later. This patient may have been of the type referred to by Goodman & Gilman (1941) in whom relatively small doses of digitalis may cause coupling, as he was taking only gr. i tab.dig.pulv. once daily.

The Effect of Treatment.

Series B.C. In one of the hospital cases of fibrillation it was possible to follow the effects of treatment, first with digitalis then with quinidine, on the response to the standard bed exercise over a period of more than a month. This patient, a man aged 34, was admitted to hospital with a history of palpitation and dyspnoea of five weeks duration. He was very

distressed and orthopnoeic on admission and the ventricular rate at rest was 130 per minute. The electrocardiogram showed typical coarse fibrillation. He was given Digoxin 0.5 mg. four times a day. The first tracing was taken three weeks after admission and the response to the standard arm-stretching exercise is given in Fig. 51, Curve A. The mean resting rate has fallen to about 110 beats per minute but it is still very variable, reaching 130 in some 5 second periods. The exercise caused a rise to a maximum of just over 140 beats per minute. The next test (Fig. 51, Curve B) was performed a fortnight later. The effects of rest and digitalis have now become apparent and the resting rate has dropped to about 70 per minute. The actual increase in rate due to the exercise is practically the same as in Curve A and there is a striking similarity in the behaviour of the two curves during exercise. They both show a peak between 15 and 20 seconds preceded by a delayed acceleration, and a second peak just before the end of exercise. Immediately after Curve B was taken the Digoxin was stopped, and two unsuccessful attempts were made to restore normal rhythm with Quinidine. Tracings were taken during both these attempts, but, as the graphs were very similar to Curve A (Fig. 51) they are not reproduced here. The next exercise test (Fig. 52, Curve C) was performed one week after Curve B

and twelve hours after the patient had completed a third course of quinidine, (four doses of 0.4 gm. quinidine sulphate at 3 hourly intervals). As can be seen from Curve C this attempt to restore normal rhythm was also unsuccessful. In this curve the immediate acceleration at the beginning of exercise is much greater than in Curves A and B and the drop in rate after exercise ends is delayed. Curve D, taken three days later immediately after a fourth unsuccessful course of quinidine, shows the same general type of response to the exercise, the ventricular rhythm still remaining totally irregular. Curve F was taken five days after Curve D and during this interval the patient had the following treatment:

First day - Tinct. Digitalis m.xx.t.i.d.

Second day - Quinidine sulph. 0.4gm. 4 hrly. for 4 doses.

Third day - No medication.

Fourth day - No medication.

Fifth day - Quinidine sulph. 0.4 gm. 4 hrly. for 2 doses.

The ventricular rhythm then suddenly became slow and regular and Curve F was recorded about an hour afterwards. The resting rate was now well within the normal limits of steadiness and the exercise caused even less increase in rate than was shown by the average curve for normal subjects (Curve E, Fig. 51), the actual increase in rate being just

half that of the normals. Part of the actual records taken before and after return to normal rhythm are shown in Fig. 53. In the latter case the rhythm can be seen to be absolutely regular apart from occasional auricular extrasystoles, one of which occurred during the exercise. The patient was then given decreasing doses of quinidine for four days. After three more days free from medication Curve G (Fig. 52) was taken. This final curve showed a greater response to the exercise than Curve F and indeed closely resembled the normal curve (Curve E). The rhythm was regular apart from two ventricular extrasystoles which occurred during the exercise.

The Postural and Exercise Components of the Step Test.

Series C. An attempt was made in four of the ambulant auricular fibrillation cases to analyse the curves taken during the step test into the 'postural' and 'exercise' components, as was done in the case of the normals and the V.D.H. cases. The sudden arbitrary variations in rate, however, made it impossible to obtain consistent results and the graphs are not reproduced here. The change from the sitting to the standing posture generally produced a slow rise in heart rate over a period of about 30 seconds in the fibrillation patients; though in one case the rate fell on standing. The only consistent feature of the 'exercise' component was the delayed acceleration already mentioned in

connexion with the total curves for exercise. This delayed acceleration will now be discussed in detail.

The Delayed Acceleration during Exercise.

Series C. In Series C, out of a total of 22 fibrillation tracings analysed, 21 showed a definite sudden increase in heart rate about half way through the exercise. The single exception was the case in which very marked and persistent digitalis coupling was present. Fig. 54 reproduces four actual tracings of fibrillation patients during exercise, showing the sudden acceleration. A normal tracing is also given for comparison. The effect was seen in both digitalized and non-digitalized patients; and with the exception of the single case mentioned above in which the dose was such as to cause coupling, the sudden onset of the acceleration was not affected by digitalis. The average delay from the beginning of exercise to the commencement of the acceleration was 12 seconds, and its average duration 25 seconds. It was pointed out above that this sudden delayed acceleration could be seen in many of the graphs of heart rate plotted at 5 second intervals during exercise in the fibrillation patients. The actual sudden increase in rate, however, was greater than would appear from these graphs because it rarely happened that the abrupt onset of the acceleration coincided with the beginning of one of the

5 second periods in which the tracings were counted.

To obtain a more accurate measure of the delayed acceleration the heart rate was counted over intervals of three seconds immediately before and immediately after its onset. In addition, a count was made over the three-second period in which the heart rate attained its maximum value. The average results for the 22 tracings showed that over the three seconds immediately following the commencement of the acceleration the heart rate had increased by 35 beats per minute or 31. , compared with the rate over the previous three-second interval. This gives the very high average acceleration of 12 beats per minute per second, and confirms the impression gained from the tracings of the abruptness of its onset. The maximum rate after the acceleration showed an increase of 48 beats per minute or 42% over the immediate pre-acceleration rate. It is to be emphasised that all this took place not at the beginning of exercise, but approximately half way through the exercise period.

It therefore seemed to be worth while to attempt to analyse the frequency response in these patients even more accurately than was possible from the kymograph tracings so far employed. It was found that with care reasonably stable photographic electrocardiograms could be obtained during the actual step test by means of an amplifier and Matthews'

oscillograph; and, on occasion, even with the string galvanometer. On these records the intervals between individual beats could readily be analysed to the nearest fiftieth of a second, and these intervals could be graphed for successive beats throughout the entire exercise.

Fig. 55 shows one such graph. The abscissa shows the individual beats reckoned from the beginning of exercise and the ordinate gives the time intervals between successive beats, converted to beats per minute for convenience in interpretation. The complete irregularity of rhythm typical of auricular fibrillation is well shown throughout the graph, but it can also be seen that a definite change takes place at the nineteenth beat after the beginning of exercise. Up to that point the heart rate swings around a mean of approximately 100 beats per minute, reaching a maximum of 130 on occasion; and the exercise has therefore had comparatively little effect. At the nineteenth beat the sudden acceleration begins and thereafter the heart rate swings about a much higher mean, approximately 150 beats per minute. In this patient the acceleration persisted for some considerable time after the end of exercise, and was interrupted by sudden brief slowings both during and after exercise. It was not possible to record these fast electrocardiograms continuously for very long periods so the

pre-exercise record was necessarily short. However, careful examination of the usual kymograph tracing (Fig. 56) taken prior to and then simultaneously with the fast electrocardiogram, made it clear that in this patient no such acceleration occurred during the resting period before exercise. Another graph of the successive intervals between beats in a different fibrillation patient is given in Fig. 57. This shows some marked differences from the previous graph. The irregularity is greater and it can be seen that very fast rates up to the maximum of 200 per minute occur in bursts while the patient is at rest before exercise has begun. This is also visible in the kymograph tracing, Fig. 58. Nevertheless, a definite acceleration occurs at the twelfth beat after the beginning of exercise and though the acceleration is interrupted by a transient fall in rate, the mean level of heart rate is higher thereafter. It was found that in general the fibrillation patients could be divided into two groups. In the first group, which comprised two-thirds of the patients, the heart rate behaved as in Figs. 55 and 56; no very fast groups of beats occurred at rest and the sudden delayed acceleration during exercise was usually very definite. In the second group, of which Figs. 57 and 58 are typical, fast beats occurred at rest and the delayed exercise acceleration, though present, was not so

clearly defined. One feature of both groups was that the heart rate, as measured by single beat intervals, had a definite maximum which was usually repeated several times. Thus in Fig. 55 this maximum was 176 beats per minute and in Fig. 57, 200 beats per minute. This was only apparent on careful beat to beat analysis and was perhaps due to the fact that the fundamental phenomenon in auricular fibrillation, the circus wave, recurs with a definite frequency. There was no clear relation between the 'coarseness' of the fibrillation waves and the occurrence of fast groups of beats at rest, however.

It was mentioned above that it was possible, with care, to obtain reasonably good electrocardiograms during exercise with the Matthews' oscillograph and sometimes with the string galvanometer. Such records were examined to see whether any marked electrocardiographic changes took place at the onset of the delayed acceleration. Examples of such electrocardiograms are shown in Figs. 60, 61, 62 and 63 but it will be more convenient to deal with the interpretation of these in the general discussion on fibrillation.

Series B.C. The delayed acceleration was also seen in most of the curves of heart rate taken during the arm-stretching exercise, but was less consistent and less well marked than in the ambulant fibrillation patients. This was probably due to the extremely mild nature of the exercise performed by the

bed patients. It is, however, clearly visible in the graphs of Figs. 46, 47 and 51.

DISCUSSION.

It is now universally accepted that the fundamental abnormality in auricular fibrillation is a wave of contraction which travels in a circular path through the auricular muscle, the 'circus movement'; and that this travelling wave supersedes the sino-auricular node as pacemaker. This theory is based on the work of Mayer (1908), Mines (1914) and Garrey (1914). The evidence was summarised by Garrey (1924) and Lewis (1925), while an exhaustive review of the literature was recently published by Rijlant (1945). One observation, however, which is difficult to explain on the theory of the single circus wave was published by Brams & Katz (1931). They showed that if auricular fibrillation was induced in the dog's heart and the two auricles were then separated from each other the fibrillation still continued in each separate chamber with very little change in the rate of the fibrillation waves.

Blumgart (1924) was the first to attempt a quantitative estimate of the effect of exercise on the heart rate in cases of fibrillation. The exercise consisted in stepping twenty times on and off a chair seventeen inches high, and the test was carried out by six controls and nine patients. Heart rates were recorded before and immediately

after the exercise by polygraph in the controls and by string galvanometer in the fibrillation cases. No records were obtained during the exercise period. He concluded that fibrillating hearts responded to a given test exercise by a disproportionate rise in ventricular rate and by a delayed return to normal. Both these findings are upheld by the present results as can be seen from Figs. 43 and 44.

Blumgart also found that digitalis in ordinary doses failed to prevent the exaggerated response to exercise as shown by the difference between pre- and post-exercise rates in the same patients before and after treatment with tinct. digitalis m.l0 t.i.d. for a month. In fact, the actual increase in rate was slightly greater under digitalis. In the present series it was not possible to compare the results for the same patients while on and off digitalis, but it can be seen from Table 11 that the actual increase in heart rate of the digitalized cases was much greater than in the normals so that here also digitalis has failed to protect completely against an exaggerated rise in rate. The actual increase, however, in the non-digitalized patients is on the average considerably greater than in patients of the same tolerance group who were receiving the drug. In general, if we consider the patients in the 'poor tolerance' group of whom four were on digitalis while three were not, all the indices except the

acceleration of the heart rate are higher in the non-digitalized group. Digitalis, therefore, while failing to abolish the exaggerated increase in rate, did diminish it to a considerable extent in my cases.

The mechanism by which digitalis slows the heart in such cases has been the subject of much investigation with rather conflicting results. Nielsen & Abdon (1937) from a survey of the earlier literature and from their own work concluded that digitalis and allied substances act on the heart muscle sensitizing it to the normal vagal tone. Abdon, Hammarskjöld & Nielsen (1938) carried this theory a stage further when they showed that digitalis sensitizes heart muscle, including that of the human heart, to acetylcholine. Boas (1931a) was of the opinion that in fibrillation the ventricular rate is governed by the balanced activity of the vagus and accelerator nerves. If vagus tone is preponderant the rate was slow and stable, whereas if the sympathetic was predominant the ventricular rate is rapid and mobile and requires larger doses of digitalis to stabilize it. The question of whether digitalis could slow the ventricular rate in fibrillation in the absence of heart failure was studied by Levy & Boas (1938). They concluded that it could do so, contrary to previous opinion. As was stated above, Blumgart (1924) found that ordinary doses of digitalis had no effect on the increase of heart rate due to exercise. Lewis (1934) was

in agreement with this, saying that though digitalis can control the rate in fibrillation patients at rest or in very mild exercise, this is rarely possible in conditions of freer exercise. On the other hand, Weinstein, Plaut & Katz (1940) demonstrated that digitalis when used in large therapeutic doses definitely lessened the ventricular acceleration due to a standard exercise test in ambulant fibrillation cases. The effect produced would therefore appear to depend rather critically on the dose of digitalis.

This is of interest in connexion with the work of Gold, Kwit, Otto & Fox (1939) who analysed the relative importance of the vagal and extra-vagal mechanisms by which digitalis slows the ventricle in fibrillation. They found that the slowing caused by small doses of digitalis (up to about 60 per cent of the full dose) could be counteracted by large doses of atropine, so that it was largely due to vagal stimulation. When full doses of digitalis were given, atropine could no longer increase the ventricular rate and the slowing was then clearly due to extravagal actions of digitalis. The main extravagal action was said to be the increase in the refractory period of the A-V conduction system. In 1941, Modell, Gold & Rothendler applied these results to the exercise acceleration of the ventricle in fibrillation patients. They concluded that in the average fibrillation

patient the exaggerated acceleration during exercise was due chiefly, if not entirely, to decrease in vagal tone. In such cases blocking the vagus by atropine accelerated the ventricles to the same maximum level as extreme physical exertion. 'Extra-vagal' digitalization with large doses prevented the exaggerated response to exercise through direct action on the A-V conducting system, in accordance with the theory. They also pointed out that the ventricular rate at rest does not indicate whether digitalis has caused slowing by the 'vagal' or the 'extravagal' mechanism, but that there are two simple ways of detecting 'extravagal' digitalization. Either two mg. of atropine may be given intravenously or the patient may be made to exercise. If neither of these procedures raises the ventricular rate to over 100 per minute, then enough digitalis has been given to cause slowing by the extravagal mechanism. Judged by the latter test, the extravagal mechanism can only have been prominent in one of the digitalized cases of the present series and there it was associated with marked coupling of beats. In this case the standard exercise only accelerated the ventricular rate from 71 to 89 beats per minute.

With regard to the effect of quinidine it was noted above that after normal rhythm had been restored by the drug the curve of heart rate during exercise closely resembled the normal one (see Figs. 52 and 53). This is in agreement with

the results of Alt, Walker & Smith (1930) though the exercise they employed was much more prolonged and the heart rate was only taken by palpation at intervals of two or four minutes.

Results given by Iliescu in Blumgart's article (1924) indicated that when the ventricular rate rises on exercise in fibrillation patients the auricular rate generally falls. This is in accordance with the usual inverse behaviour of the auricular and ventricular rates in this condition. In the present series it was impossible to determine the fibrillation rate of the auricles during the exercise with sufficient accuracy to give valid results, but the pre-exercise fibrillation rates could be counted from the electrocardiograms in eight cases and varied from 375 to 600 per minute. An attempt was made to correlate the pre-exercise fibrillation rate with the amount of the delayed acceleration of ventricular rate during exercise in these cases. Taking three-second periods immediately before and after the onset of delayed acceleration, it was found that the slower the fibrillation rate the greater was the actual increase in ventricular rate, as the following table shows:

Pre-exercise fibrillation
rate.
(oscillations per minute).

Actual increase in
ventricular rate at onset of
delayed acceleration.
(beats per minute).

600	20
500	26
500	30
444	30
420	36
408	40
408	46
375	52

This presumably indicates that when the vagal inhibition is lifted during exercise the ventricular rate can rise relatively more to meet a low auricular rate than a high one, as might be expected from the general theory of transmission from auricles to ventricles in fibrillation.

Further studies on the effect of exercise in fibrillation cases were carried out by Boas (1929) and Boas & Goldschmidt (1930), using the cardi tachometer. No quantitative data were given, but the exaggerated increase of heart rate on exercise described by Blumgart (1924) was confirmed. They found that their cases could be divided into two groups, those with labile and those with stable ventricular rates, corresponding to those with highly strung

and phlegmatic temperaments respectively. Harris & Lipkin (1931) investigated the effect of mild exercise on the cardiac output of six patients with auricular fibrillation. The increase of stroke-volume during exercise was significantly depressed compared with six normal subjects, but a high percentage of oxygen utilization was a characteristic feature of fibrillation. Katz & Feil (1923) had previously shown that in fibrillation the duration of ventricular systole was shorter than in normals at corresponding rates. Several observers have shown that in auricular fibrillation there is a reduction of 'physical efficiency' as judged by various tests. Thus Hunt & Pembrey (1921) using their 'pulse ratio' test found three cases to be only 31% efficient, and Hewlett (1924) found that the average vital capacity of fibrillation patients was 62% of the normal. It is of interest in this connexion that Jones & Schlapp (1945) showed that the induction of auricular fibrillation reduces the work capacity of the cat heart by approximately one-third. In spite of this, Harrison (1939) has pointed out that the onset of auricular fibrillation is often an asset rather than a liability to a failing heart, chiefly because digitalis can act more effectively in the presence of fibrillation. According to Altschule (1938) digitalis produces a definite increase in

the cardiac output of such patients.

One of the most interesting points about auricular fibrillation is that though the normal pacemaker, through which the nervous control of the heart rate usually operates, is superseded by a rapid and irregular 'circus movement' the ventricle can still respond to exercise and even to the phases of respiration. The mechanism by which such alterations of ventricular rate may be brought about during fibrillation has been investigated by many workers. It is known that in the normal heart the chief factor in the acceleration of exercise is the withdrawal of vagus restraint. Consequently the effect of the vagus on the fibrillating heart is of particular interest. As regards the auricle, Winterberg (1907) and Rothberger & Winterberg (1914) showed that stimulation of the vagus caused the fibrillary twitchings to become finer and more rapid. This was confirmed by Lewis, Drury & Bulger (1921) who worked out the mechanism in terms of the changes in refractory period (reduction) and in rate of conduction (increase), as they affected the circus movement. Similar results for auricular flutter were reported by Wilson (1924). With reference to the effects of vagal stimulation on the ventricular rate in auricular fibrillation, Kronecker & Spallitta (1905), Phillips (1905) and Robinson (1913a and 1916) showed that the ventricle was slowed during

experimental fibrillation in the dog when the vagus was stimulated. Lewis (1909) proved that the slowing was due to an increase in heart block. In 1914, Robinson reported a case of auricular fibrillation in the human subject in which pressure on the vagus slowed the ventricular rate; and Crawford (1923) found that cases of fibrillation showed a greater response to atropine than normal subjects. Lewis (1925) summarised the position as follows: "If we examine all the known ways of reducing the ventricular rate while the auricles fibrillate, we find that heart-block may always be ascribed as the cause". It has long been known that the vagus can affect the A-V node and the bundle of His (Herz & Goodhart 1908; Wilson 1915a and b) and recently Jourdan, Froment, Gallavardin & Baud (1945) showed that atropine increased the A-V nodal rhythm in dogs in which the S-A node had been destroyed; while Miller and Perelman (1945) found that change of posture may have an effect on the human heart rate even when the S-A node is out of action.

According to Kilgore (1919) a number of fibrillation cases show a fairly consistent tendency to have shorter intervals between the heart-beats during late expiration or early inspiration than during the opposite part of the respiratory cycle, that is, the reverse of ordinary sinus arrhythmia. An attempt was made to see whether this effect

was readily visible with the present method of recording by getting the fibrillation patients to breathe deeply. No consistent results were obtained as can be seen from Fig. 59 which shows two typical records. This is not surprising as Kilgore was only able to demonstrate the phenomenon by detailed analysis and statistical treatment of a large number of pulse curves.

From all the above work it may be supposed that during exercise in auricular fibrillation the vagus tone is diminished causing a fall in fibrillation rate and an improvement in conduction through the specialized tissues of the heart. Both factors will permit the ventricles to respond to a higher proportion of auricular impulses. While this accounts adequately for the general rise in ventricular rate it does not explain the delayed acceleration which is such a conspicuous feature of the present series. It will be seen from Fig. 54 that during the delayed acceleration the rhythm often appears to become more regular and it was thought possible that the sudden acceleration might be due to the fibrillation having changed temporarily to flutter, in which the ventricle could more easily follow the high auricular rate. Robinson (1913b) and Wiggers (1923) were of the opinion that vagus stimulation could convert auricular flutter into fibrillation, and Altschule (1945) showed that

there was a definite correlation between excessive vagal activity and the onset of fibrillation in man. Therefore the inhibition of vagal tone during exercise might possibly have the reverse effect and convert fibrillation into flutter by increasing the refractory phase of auricular tissue. Miller (1942a) reported a case in which atropine converted fibrillation to flutter in man. In order to test this theory electrocardiograms were taken during the period of delayed acceleration, and some examples are shown in Figs. 60 - 63. The actual onset of the delayed acceleration can be seen in Fig. 60 Curve E, and in Fig. 61 Curves A and B. There is no evidence in any of these records of a change from fibrillation to flutter. The same negative result is found in Fig. 62 Curve B, and Fig. 63 Curve B taken immediately after the end of exercise while the acceleration was still present. Moreover, if the intervals between beats are carefully measured during the acceleration they are found to vary in spite of a naked-eye appearance of regularity. This fact is also brought out in Figs. 55 and 57 and is an additional argument against a change to flutter.

Another possibility was that since digitalis acts partly through vagal stimulation it might be supposed that in digitalized patients this effect would oppose the withdrawal of vagal restraint by which exercise normally

accelerates the heart. If this 'holding down' effect of digitalis took some time to overcome but then suddenly gave way, the delayed acceleration would result. This explanation, however, is unlikely because the delayed acceleration occurs equally in non-digitalized patients and no definite correlation could be obtained between the dosage of digitalis and the time of onset of the acceleration.

Boas (1929) suggested that in the 'labile' type of fibrillation patient the sympathetic nerves might play a dominant part in producing ventricular acceleration. As sympathetic effects on the heart are characterised by a considerable latent period this might have accounted for the delayed acceleration. The results of Modell, Gold & Rothendler (1941), however, are definitely against Boas' theory, and render it improbable that direct sympathetic stimulation has any important role in producing the exercise acceleration of fibrillation patients. The latent period (about 12 seconds) is such that sympathetic acceleration of the heart by means of adrenaline liberated from the suprarenals cannot be excluded, however,

Apart from this possibility it would seem, therefore, that the delayed acceleration is most probably mediated through the vagus nerves. It might be argued that though in normal rhythm the action of the vagus on the S-A node is almost instantaneous, in fibrillation, where it must operate

mainly through effects on the conducting system, there might be a considerable delay. There is little published information on this point, but from examination of the records illustrating the articles of Phillips (1905), Lewis (1909), Robinson (1916) and Lewis, Drury & Bulger (1921) it would appear that when the vagi are stimulated during auricular fibrillation in the dog's heart slowing of the ventricles occurs after a delay of only about one second. Lewis (1925, p. 348) published an electrocardiogram in which pressure on the carotid sheath in man slowed the ventricle during auricular fibrillation after a latent period of just over one second. Hill (1933) on compression of the exposed vagus nerve in man during normal rhythm found the latent period to be of the order of two to three seconds. This last observation is difficult to reconcile with the very brief latent period (less than one cardiac cycle) observed when the normal heart accelerates at the beginning of exercise or slows during a sudden fright, and it would seem that the results obtained by such abnormal procedures may not be strictly applicable to similar events occurring in the intact organism. It is therefore quite possible that there may be a delay in the lifting of the normal vagal restraint during exercise under the special conditions of auricular fibrillation.

A final possibility is that the Bainbridge reflex plays a part in causing the delayed acceleration, though the work of Sharpey-Schafer & Wallace (1942) seems to show that a rise of venous pressure in man does not always cause an increase in heart rate. Normally, this reflex would appear to have a high threshold in man, but the increased resting venous pressure often found in fibrillation might be expected to counteract this and render the reflex more effective.

The rise in venous pressure at the beginning of exercise is almost certainly not instantaneous, though there seems to be very little information on this point. In Hooker's experiments on human subjects readings were only taken at intervals of one minute (Hooker 1911), but McCrea, Eyster & Meek (1928) found that the venous pressure usually rose by about 5 cm. in the first half-minute of exercise on a bicycle ergometer, and that the heart rate rose rather more slowly than the venous pressure. In animal experiments Bainbridge (1915) found the delay before the heart began to accelerate to be proportional to the rate of injection of saline into the veins, and Sassa & Miyazaki (1920) showed that cardiac acceleration only occurred if the distension of the right auricle lasted for several seconds, a very brief distension produced no effect. The average latent period in

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their experiments was about four seconds. Anrep & Segall (1926a) working with the innervated heart-lung preparation found that when the venous pressure was raised the heart began to accelerate a few seconds after the cardiac output was increased. It is probable therefore that in man there will be a delay of several seconds before the venous pressure rises sufficiently to trigger the Bainbridge reflex.

It is possible that several of the factors mentioned above may combine to produce the delayed acceleration under the conditions of diminished sensitivity prevailing when the S-A node has ceased to be the pacemaker; and in this connexion it is to be noted that the heart block cases, in whom the ventricles were also not under the control of the S-A node, showed a similar delay in the onset of cardiac acceleration during exercise.

SUMMARY.

The effect of the standard step-test on the heart rate of ambulant cardiac patients was investigated. Records were also taken of the heart rate in bed cardiacs during a brief standard arm-stretching exercise.

The following conditions were studied:

- (a) Valvular disease of the heart. These patients showed very little anticipatory increase in heart rate before exercise began, and the general level of maximum rate reached during the step-test was slightly less than in the normal subjects. There was no correlation between the clinical exercise tolerance and any heart rate index, except the return to normal. The effect of treatment on the bed cardiacs was investigated and it was shown that as treatment becomes effective the curve of heart rate becomes generally lower, though in some cases the pattern of cardiac acceleration is changed.
- (b) Angina pectoris and (c) Coronary thrombosis. These conditions were characterized especially by a very low maximum rate during exercise.
- (d) Effort syndrome. The main difference between these patients and the normals was in their high resting heart rates.

- (e) Heart block. In all three cases the mild exercise did produce some cardiac acceleration which was delayed compared to the normal. The pre-exercise emotional increase in heart rate was absent in these patients.
- (f) Thyrotoxicosis. One patient showed typical tachycardia at rest and a small increase in heart rate during exercise before treatment. After thyroidectomy the response to exercise closely resembled the normal. The other patient gave atypical results, both as regards B.M.R. and heart rate response to exercise.
- (g) Auricular fibrillation. In this condition the maximum heart rates during exercise were much higher than in the normals or the V.D.H. patients. In several cases there was a brief fall in rate at the beginning of exercise. Almost all the ambulant fibrillation cases showed a characteristic delayed acceleration of the heart, beginning about half way through the exercise period. Various theories are put forward to explain this phenomenon, which does not appear to have been described before.

In all cases the previous literature is discussed in relation to the present findings.

"One of a gigantic size, and of an athletic habit hath not naturally the same kind of pulse with a dwarf;.... and we often meet with people, seemingly of the same habit and constitution, who have nevertheless pulses of different kinds; from which it appears of what advantage it is to be acquainted with the constitutions of people in health, to be able to judge with greater certainty of the nature of diseases."

Stedman (1769) p. 29.

CHAPTER 11.

THE RESPONSE OF THE HEART RATE TO EFFORT AS AN INDEX OF
PHYSICAL FITNESS.

(Series S.T.C.).

The possibility that some of the heart rate indices might be correlated with physical fitness was mentioned in Chapter 5, when the literature on cardiac tolerance tests was reviewed. In all the series dealt with up to the present no such correlation could be attempted, as an independent estimate of the subjects' fitness could not be obtained. During the war, however, an opportunity arose to perform a cardiac tolerance test on volunteers from the Senior Training Corps whose fitness had been estimated independently during their tests for Certificates "A" and "B".

METHOD.

Forty-five students volunteered for the test. Their ages ranged from 17 to 19 years. As the volunteers were all in reasonably good health, the grading was simply done round an average fitness. It was considered that the standard exercise test as used with Series A, while suited to its purpose of comparing normals with ambulant cardiac patients, would be too mild to bring out minor differences in fitness among a group of healthy males. For the present purpose, therefore, the standard test was modified and its severity increased by making the subjects climb the two steps ten times carrying a load of 30 kg. The rate of stepping was

96 per minute, and the exercise was started from the sitting posture. The modified test thus differed from the standard test of 5 ascents with no load at 96 steps per minute only in the load carried and in the increased number of ascents. The average total work done during the modified test was approximately three times that of the standard test; 492 kg.m. as compared with 173 kg.m. The mean rate of working in the modified test was 742 kg.m. per minute compared with the 432 kg.m. per minute of the standard test.

RESULTS.

General. The results for the various heart rate indices are given in Table 12. Compared with the results for Series A in Table 1 the indices here are practically all higher, as would be expected from the increased rate of working. An apparent exception is the mean acceleration, which has decreased; but owing to the method of calculating this index a decrease naturally results from the prolongation of the exercise. The coefficients of variation are in fairly good agreement with those of Table 1. For comparison with the milder exercise of Series A the coefficient of correlation between post-exercise rate and maximum rate was calculated with the following result:

Coefficient of correlation = + 0.83.

Corrected standard error = 0.15.

It is interesting that the correlation coefficient is exactly the same as that for Series A (males) and shows that the severer exercise did not alter the very significant degree of correlation between the two indices. The regression equation for Series S.T.C. was:

$$\text{Maximum Rate} = 0.664 \text{ Post-exercise Rate} + 72.$$

The very high initial rate in the S.T.C. series (93.3 beats per minute) was again due to the anticipation of exercising with a load. As was pointed out in Chapter 9 this great preliminary increase is peculiar to load-carrying experiments. It was probably enhanced in the present instance because most of the subjects were quite unused to such procedures.

Fig. 64 shows a graph of the mean heart rates in 5 second periods during the exercise. Apart from the higher initial rate it is, for the first 20 seconds of exercise, in good agreement with the similar graph in the load series for 32.2 kg. load (Fig. 20). As before, the postural component has been completely swamped by the acceleration due to the load carrying. Over the last ten seconds of exercise there is no increase in heart rate, indicating that the "steady state" has been reached. The deceleration after exercise is remarkably uniform, though the curve must flatten out very considerably beyond the period actually graphed as the average time of complete return to the initial rate was 81

seconds after the end of exercise (Table 12).

The Correlation of Indices with Physical Fitness.

As a preliminary, an estimate of the fitness of each subject was obtained from the Physical Training Corps Instructor who had observed them carefully during all their tests for Certificates "A" and "B". The subjects were classed as "Above Average", "Average" or "Below Average". This classification could only be applied to 41 subjects and only these were included in the preliminary survey. The mean values for initial rate, maximum rate, actual increase and percentage increase of heart rate in the tolerance test were then calculated for each class. The results were given in Table 13, which shows that there was very little difference between the "Above Average" and "Average" groups but that the "Below Average" group had a very much higher mean initial rate and maximum rate, though the actual increase was practically the same for all groups. The marked fall in percentage increase in the "Below Average" subjects was of course due to their high initial rate. It was then decided to attempt actual correlations between some of the heart rate indices and the physical fitness. It was therefore necessary to give a numerical value to the fitness of each subject and this was carried out as follows. The Certificates "A" and "B" Physical Efficiency Standard consisted of fourteen tests,

including sprinting, running, walking and climbing. For each test successfully passed at the first attempt one mark was allotted. The Physical Instructor's estimate was then taken and three marks were given for "above average", two marks for "average" and one mark for "below average".

Finally, the most strenuous test in the course was separately marked. This consisted in running and walking for 10 miles in full kit in under two hours. The marking was as follows:

Completing run in 1 hr.30 min.to 1 hr.35 min.	- 6 marks
" " " 1 hr.35 min.to 1 hr.40 min.	- 5 "
" " " 1 hr.40 min.to 1 hr.45 min.	- 4 "
" " " 1 hr.45 min.to 1 hr.50 min.	- 3 "
" " " 1 hr.50 min.to 1 hr.55 min.	- 2 "
" " " 1 hr.55 min. to 2 hours	- 1 mark.

The maximum number of marks possible was thus 23; 14 for passing all tests at first attempt, 3 for "above average" instructor's estimate, and 6 for completing the 10 mile run in $1\frac{1}{2}$ hours. The above data were only completely obtainable for 34 of the subjects and they alone were used in the attempts at correlation. The range of physical fitness was from 11 to 22 on the above scale. It was of course realised that this method of marking was purely arbitrary, but it was to be expected that it would give some measure of a subject's physical capabilities.

The first correlation attempted was between physical fitness and the maximum rate in the cardiac tolerance test. The coefficient of correlation $r = -0.614$ with a corrected standard error of 0.174. As the correlation coefficient was more than 3.5 times the standard error there was a significant correlation between fitness and maximum rate, the negative sign indicating that the higher the fitness the lower the maximum rate.

A significant though lesser degree of correlation was found between physical fitness and the post-exercise rate, the correlation coefficient $r = -0.503$ with a corrected standard error of 0.174.

There was no significant correlation between fitness and absolute increase in heart rate or between fitness and acceleration of heart rate, the coefficients being:

Absolute increase and fitness, $r = -0.099$; S.E. = 0.174.

Acceleration and fitness, $r = +0.035$; S.E. = 0.174.

When group correlation tables were constructed it was obvious by inspection that there was also no correlation between fitness and initial rate, percentage increase on initial rate, or time of return to normal.

Sinus Arrhythmia.

For comparison with the results in Series A the occurrence of sinus arrhythmia was determined in the S.T.C. series. The results were as follows:

Sinus Arrhythmia	Series S.T.C. (percentage)	Series A (percentage)
Absent	9.0%	9.3%
Slight	20.0%	29.3%
Marked	44.0%	54.7%
Very Marked	27.0%	6.7%

The respiratory arrhythmia was thus even more marked in the S.T.C. series than in the original series. There was no correlation between the degree of sinus arrhythmia shown by a subject and his physical fitness index.

DISCUSSION.

The results of a number of fitness tests were discussed in Chapter 5, but a few further points may be mentioned here. In view of the fact that training generally decreases the resting heart rate (Dawson, 1919; Cotton, 1932) it is curious that there was no correlation between fitness and the initial rate. This is however in agreement with the results of Brouha & Heath (1943) and Gallagher & Brouha (1944) who found no satisfactory relation between the resting heart rate and the individual's ability to perform work. As regards the maximum rate, Taylor (1944)

found that only during the exercise did the heart rate curves sort themselves out to indicate the fitness groups correctly. The present results are in agreement with this in so far as the correlation with the maximum rate during exercise is greater than with the post-exercise rate; though a significant correlation was here obtained with the latter. The fact that no correlation could be obtained between fitness and the acceleration of the heart rate is in agreement with Dill & Brouha (1937) who found that athletic training did not increase the speed with which the heart accelerates at the beginning of work. The significant relationship between fitness and the post-exercise rate might have been expected from the results of Cogswell, Henderson & Berryman (1946) who showed that the latter decreased with training, and from those of Taylor & Brozek (1944) who were of the opinion that the heart rate after a standard amount of work is probably the most useful single criterion of fitness. On the other hand, Woolham & Honeyburn (1927) found no correlation between fitness and the immediate post-exercise rate in schoolboys. In view of the large number of fitness tests which depend on the time of return to normal (e.g. Johnson, Brouha & Darling, 1942 and Johnson & Robinson, 1943) it is surprising that no correlation was obtained between this index and fitness in

the present series. The general conclusion from the present experiments was that when a moderate exercise of short duration is employed as the test, the only heart rate indices which may be of value in estimating physical fitness are the maximum rate during exercise and the immediate post-exercise rate.

It was found that there was no significant correlation between fitness and the following heart rate indices: acceleration, absolute maximum rate, percentage increase and time of return to normal. These findings are discussed in relation to the results of other workers.

SUMMARY.

Forty-five students, whose physical fitness had been independently estimated, performed a modified version of the standard step test (10 ascents carrying a load of 30 kg.). The values and variability of the heart rate indices are compared with those of Series A (normal males). A significant correlation was found between physical fitness (on an arbitrary scale) and the maximum heart rate during exercise. A significant, though less complete, correlation was obtained between fitness and the post-exercise rate. There was no significant correlation between fitness and the following heart rate indices: acceleration, absolute increase, initial rate, percentage increase and time of return to normal. These findings are discussed in relation to the results of other workers.

"On this subject, the opinion of those, whose profession it is to train men to the performance of great muscular feats, when they speak the truth, is of much more consequence than that of any medical man."

Robert Knox (1815), p. 62.

CHAPTER 12.

THE HEART RATE OF A MARATHON RUNNER DURING EXERCISE.

A great deal has been written about the effect of athletic training on the heart rate and it is not proposed to deal with the progressive effects of such training on individual subjects here. The opportunity, however, occurred of obtaining as subject a long-distance runner who had once been of Olympic standard; and it was thought that it might be of interest to compare his reactions to the standard exercise with those of the ordinary medical students. In addition, it was found possible to record his heart beats continuously throughout a 6 mile run on a motor-driven treadmill.

The subject was D.M. Wright, a well-known Scottish amateur Marathon runner. He won the Marathon at the British Empire Games in 1930 and was fourth in this race at the 1932 Olympic Games. From 1935 to 1942 he did not run in competitions but in the latter year, at the age of 46, he restarted competitive running. Since then he has run in 41 races and won 35, setting up several local records. The present tracings were recorded in 1943 when Wright was 47 years of age.

There are two points of medical interest in connexion with Wright as a Marathon runner. First, it is

unusual for a man aged 47 to be able to compete successfully against younger men in endurance events. Second, it was found that Wright had a well marked apical systolic murmur. The murmur was not conducted and was almost certainly 'functional'; though it was definitely not associated with a tachycardia, as such murmurs usually are (Luisada & Mautner, 1943). Herxheimer (1932) and Jokl & Suzman (1940) have recorded instances of organic valvular disease in successful marathon runners, however. Wright's mean resting blood pressure was found to be 110/70, a remarkably low value for a man of 47.

METHOD:

The standard two-step test was performed exactly as in Series A (5 ascents at 96 steps per minute with no load).

The six mile run was carried out on a motor driven treadmill similar to that described by Benedict & Murschauser (1915). The machine was set for level running and the distance traversed and the average speed were obtained at intervals from a revolution counter connected to one of the rollers. Heart beats were recorded electrically on a kymograph by the method described in Chapter 3; the only modification being that it was found advisable to earth the subject through a pad applied to the small of the back, as otherwise the static electricity generated by the friction

of the running shoes on the endless belt interfered with the clear recording of the heart beats. Respiration was recorded by an ordinary stethograph and tambour.

The subject stated that conditions were very similar to those obtaining during an actual race, except that there was a tendency to overestimate the distance run. This might be accounted for by some slip between the shoes and the endless track.

RESULTS:

(a) The Standard Two-Step Test.

There were two objects in view here, first to compare the response of this trained athlete with that of the average medical student of Series A; and second, to see whether a run on the treadmill caused any marked alteration in the response to the standard two-step test.

Fig. 65 shows a kymograph tracing of Wright's performance in the two-step test. Before exercise begins the heart beats display the slow regular rhythm typical of the trained long-distance runner, and the heart rate remains comparatively low throughout the brief exercise. There is no evidence of sinus arrhythmia. In general, it was found that Wright's initial and maximum rates were much lower than the average for Series A and that his actual increase in rate was also lower. These points are brought out in Fig. 66 in

which graphs of the heart rate during the standard exercise are shown. Curve A is the mean heart rate for the male medical students of Series A, while Curve B shows a typical result for Wright before a run on the treadmill. It will be seen that apart from the points mentioned above, Wright's curve closely resembles the normal one. Curve C shows Wright's response to the same standard step-test ten minutes after the end of a short run (about half a mile) on the treadmill. Compared with Curve B, taken before the run, the general level of Curve C is lower; which would seem to indicate that the brief run had a beneficial effect on the response to the step test. To investigate this point further, when Wright carried out the full run of 5.9 miles on the treadmill some six weeks later he was asked to perform the standard two-step test ten minutes before and again ten minutes after the run. The results are shown in Fig. 67, Curve A being the response of the heart rate before the run and Curve B the response ten minutes after the run. As in the previous figure the initial and maximum rates are definitely lower in the curve taken after the run, but the actual increase in rate due to the step test is practically identical in the two curves. After the 6 mile run the deceleration following the step test is slower, but this may be due to the considerable difference in the initial rates. The consistently lower initial and maximum rates in the step test after running

are probably due to a relaxation of psychological tension, but they would also seem to indicate that a run of six miles was insufficient to cause any signs of circulatory strain in this subject.

(b) The Six Mile Run.

The kymograph tracing of the start of the run is reproduced in Fig. 68. The steady acceleration of the heart rate, beginning immediately the treadmill was started and continuing for about one minute, is clearly visible. The respiratory tracing shows that very soon after the start the breathing settled down into a definite though somewhat irregular rhythm, the normal breaths being interrupted at intervals by single deep respirations. This continued throughout the entire run. The subject felt that on this occasion he took his deep breaths a little more often than usual because of the slight constriction of the electrode and stethograph straps round his chest. Graphs of the heart rate and respiration rate counted in 30 second periods throughout the run are shown in Fig. 69. The speed of running in miles per hour is also given. One minute after the start the heart rate had increased from the resting (standing) rate of 60 to 116 per minute, and continued to fluctuate around this level. At the same time the respiration rate increased from the rather high resting level of 25 to about 40 per minute. The speed at this time was about 6 m.p.h.

At the end of 5 minutes the subject increased speed to 7.3 m.p.h. and the heart and respiration rates rose accordingly. Shortly afterwards Wright reported that he had got his 'second wind' and thereafter the heart rate slowly fell until about half way through the run. At this point he again increased speed slightly and the heart rate rose, but in spite of a further small increase in speed to nearly 8 m.p.h. near the end of the run it then remained fairly constant until the finish. No definite change in respiration occurred at or following 'second wind', but in general the fluctuations of respiratory rate were greater than those of heart rate. At the end of the run the respiratory rate dropped almost immediately to the pre-exercise level; whereas the heart rate, after an immediate steep fall, returned more slowly. It is interesting that a man of 47 could run a distance of 6 miles at an average speed of 7.5 m.p.h. without his heart rate rising above 130 per minute at any point.

(c) The Heart Rate during the Start of a Run.

A more detailed picture of the rise of heart rate at the beginning of the run was obtained by analysing the record in 5 second intervals during the first two minutes of exercise. The result is shown in Curve A of Fig. 70. It will be seen that the heart rate increased rapidly for the

first fifteen seconds and then more slowly until a fairly steady rate was reached after 50 seconds.

In order to find out whether there was much variation in the cardiac acceleration at the start of a race, a further series of tests were made as follows. At intervals of half an hour Wright made three separate starts, but the run was stopped after about two minutes in each case. The results are graphed as Curves B, C and D in Fig. 70. It will be seen that in spite of small variations in the speed of running there was remarkable uniformity in the curves. This is all the more noteworthy because almost a year elapsed between Curve A and Curves B, C and D.

DISCUSSION:

The relatively slow resting heart rate of trained athletes has been described by many writers. One of the first to study it in detail was Florence Buchanan (1909) who concluded that after training the slow rate was due to groups of longer diastolic pauses scattered irregularly amongst normal ones of the same length as before training. A similar phenomenon had been noted after exercise in trained men by Pembrey & Todd (1908). No evidence of such irregularity was seen in the present case. Buytendijk (1928) found that the mean resting heart rate of the Marathon runners in the Olympic Games of that year was 58 beats per minute, which is in

excellent agreement with the present results. Very low resting rates (below 50 per minute) have been recorded by Cotton (1932) and White (1942) in highly trained athletes. The general view is that the bradycardia of trained men is due to an increase of the normal vagus tone and the evidence for this was reviewed by Steinhaus (1933); though recently Müller (1942) concluded that peripheral changes, particularly in the muscles, may play a larger part than has hitherto been realised. A corollary of Müller's work is that training must be very specific for each form of sport, and evidence in favour of this has been provided by Bøje (1944) and Brouha (1945).

Dill & Brouha (1937) using a treadmill, found that training did not increase the speed with which the heart accelerated at the beginning of work. This was confirmed in the present case for the step test as can be seen from the rising slope of the curves in Figs. 66 and 67. With regard to the actual increase in heart rate during exercise, Hunt & Pembrey (1921) and Jung, Cisler & Müller (1946) recorded smaller increases in heart rate for a given exercise in trained men than in untrained men; and Taylor, Erickson, Henschel & Keys (1945) obtained the reverse effect after three weeks of bed rest in normal subjects. These results were confirmed in Wright's case for the step test, and would also be in

accordance with the very low maximum rate recorded during the six mile run. As was pointed out above, one of the most striking features of this run was the steadiness of the heart rate. This is in agreement with the suggestion of Dill & Brouha (1937) that the ability to maintain a 'steady state' for long periods is a characteristic of the trained man. Their study included records from the veteran runner De Mar, then aged 47, and it is interesting that his curve of heart rate for moderate exercise on a treadmill closely resembles that of Wright, both qualitatively and quantitatively. Nylin (1945) noted that Hägg, the long distance runner, developed marked sinus arrhythmia after exercise, but in Wright's case no respiratory arrhythmia was seen at any time.

In general, these results confirm for the circulatory system the finding of Benedict & Cathcart (1913) that the average efficiency of the trained subject is greater than that of the ordinary man.

SUMMARY.

A highly trained long-distance runner performed the standard exercise and the results were compared with those of the normal subjects. He also ran six miles on a treadmill, the heart beats and respiration being recorded continuously. The maximum heart rate attained during the run was 130 beats per minute, and in general the heart rate remained very steady. The curve of acceleration of the heart rate at the beginning of a run was also found to vary very little on successive occasions.

The literature on the response of the heart rate to exercise in trained men is discussed in relation to the present results.

"A strong extension of the Legs and Arms by the Power of the Will, has quickened the Pulse twenty beats in a minute, and at the same time made it so low, that it could scarcely be felt."

Bryan Robinson (1732), p. 177.

CHAPTER 13.

THE RESPONSE OF THE HEART RATE TO STATIC EFFORT.

The exercises dealt with up to this point have been predominantly of the dynamic variety, involving the rapid brief contraction and relaxation of muscles. The main characteristic of the static type of effort is sustained contraction of muscle groups, and there is some evidence that the circulatory adjustments to this kind of effort are different from those of the more usual dynamic type.

To investigate whether such a difference exists in the frequency response of the heart during a brief static effort comparable to the step test, preliminary experiments were carried out on a group of eight healthy male students.

METHOD:

Heart beats and respiration were recorded on a kymograph as in the step test experiments. The subject sat on a chair and relaxed as completely as possible for five minutes. At the end of this time the recording apparatus was started and thirty seconds later the order to "pull" was given. Thereupon the subject grasped the handles of two "Terry" springs which were suspended in the horizontal position immediately in front of him, and pulled them out as rapidly as possible to a standard distance. This was indicated by pointers and was equivalent to a pull of 7.5 kg. with each arm.

The subject then kept the pointers at the 7.5 kg. mark for twenty seconds until the order to "let go" was given, when he returned the springs to zero and again relaxed. The back was supported throughout the experiment to ensure that only the arm and shoulder muscles could be employed. The heart rate was counted from the kymograph record in five second periods before, during and after the effort; the moment of beginning the pull being taken as the zero point.

RESULTS:

The type of tracing obtained under these conditions was remarkably uniform, and a typical record is shown in Fig. 71. It will be seen that there was an immediate acceleration of the heart rate at the beginning of the pull, but after about ten seconds this gave way to a marked slowing which lasted until the springs were released when a second marked acceleration occurred. The respiratory record shows that there was no tendency to fix the chest during the static effort; after the initial irregularity in breathing had passed off the respirations became regular and somewhat deeper than during rest.

When the mean heart rates during successive five second periods were graphed for the whole series, the initial acceleration, subsequent slowing and release acceleration were well shown (Fig. 72). The shape of the curve during the

actual pull was not unlike that of the average response to the step test shown in Fig. 13, in that both show a drop in heart rate during the actual effort. In the case of the step test this proved to be due to the withdrawal of the postural component of the work and it seemed probable that a dual mechanism was also showing itself in the heart rate curve of the static effort.

It was of course realized that when the subject pulled out the springs, maintained them in tension and then released them, both dynamic and static effort were involved; and that with such a brief holding time the proportion of dynamic to static component was probably fairly high. Further, the dynamic portion of the effort could be split up into positive work while the spring was being pulled out and negative work while the spring was being released. The twin accelerations were thus almost certainly due to the dynamic muscular movements involved in pulling out the springs and in returning them to rest. In order to check this and to see whether it would be possible to separate the effects of the static and dynamic components of such a brief mixed effort on the heart rate, a further series of experiments was carried out. The technique was similar to that described in Chapter 7 for the analysis of the postural and exercise components of the step test.

METHOD:

The experiments were carried out on a series of fifteen male students. Each subject first performed the static effort test exactly as in the preliminary series, pulling out the springs to a load of 7.5 kg., holding them there for 20 seconds and then releasing them. The subject then remained at rest for ten minutes, by which time the heart rate and respirations had returned to normal. The experiment was then repeated exactly as before, except that this time the distal ends of the springs were uncoupled from the wall and the springs were suspended from a trapeze so that they could swing easily forward with negligible pull. Thus only the dynamic effort of flexing the arms during the pull and of extending them during release was required. The position of the arms and the amount of flexion needed remained exactly as in the previous test. This might be called the "Sham Static Effort" experiment. The heart beats were recorded and counted as in the preliminary series, but a stethograph was placed round the abdomen as well as round the chest in order to see whether there was any marked fixation of the abdominal muscles which might play a part in the heart rate changes.

RESULTS:

The type of tracing obtained was again remarkably uniform. Typical tracings for the two experiments in the

same subject are reproduced in Figs. 73 and 74. It will be seen that no serious interruption of either thoracic or abdominal respiratory movements occurred in either case. The average heart rates during successive five second periods were graphed for the whole series of fifteen subjects, (Fig. 75), the upper curve showing the heart rates during the 7.5 kg. pull and the lower curve the heart rates during the "sham" static effort. The upper curve thus represents the dynamic component plus a static effort of 7.5 kg. while the lower represents dynamic effort plus the very slight static effort involved in keeping the arms bent for twenty seconds against negligible resistance. Both curves show a well marked initial acceleration, subsequent slowing and release acceleration.

If the effects of the static and dynamic components on the heart rate are additive, as was found to be the case with the postural and exercise components of the step test, it should be possible to obtain the response of the heart rate to the purely static component by subtracting the heart rates in each 5 second period of the lower curve from those of the upper. When this was done the graph shown in Fig. 76 was obtained. The curve rises in almost a straight line to a maximum at the end of the effort and then falls again at practically the same even rate. If this slow steady acceleration does indeed represent the response to the 7.5 kg.

static component alone it would be expected that if the same pull of 7.5 kg. were maintained for a much longer period the heart rate would show a steady increase, since the static component would continue to act after the effect of the initial dynamic component had passed off. Fig. 77 shows this to be the case for three subjects pulling on the springs until they could no longer maintain the pointers steady on the 7.5 kg. marks. The heart rates were counted in 5 second intervals throughout and all three curves show the initial acceleration and subsequent fall due to the dynamic component, but after this has passed off the heart rate does rise steadily. Immediately following the end of the prolonged static effort there is a very sudden drop in the heart rate in all cases. When this immediate post-exercise deceleration was compared with that following the step test in Series D in which both the duration of exercise and the heart rate at the end of exercise are reasonably comparable, it was found that the drop in rate was greater after the static effort, at least during the first ten seconds of the recovery period.

DISCUSSION:

It would seem, therefore, that the above results can be adequately accounted for by supposing that the dynamic and static components of the effort each affect the heart rate and that the effects are additive. In view of the work of Bowen (1903) it is not surprising that a slight muscular movement

such as flexing the arms should cause a well marked cardiac acceleration. Cathcart & Stevenson (1922) found that negative work is performed with a lower energy expenditure than positive work and this may be reflected in the graphs of Figs. 72 and 75 which show that the initial acceleration was greater than the release acceleration.

As regards the differences between static and dynamic effort, the experiments of Lindhard (1920 and 1923) seemed to indicate that static effort is in the main anaerobic, and that the statically contracted muscles impede the bloodstream mechanically. The static effort in his experiments consisted in hanging by the hands from a horizontal beam. Cathcart, Bedale & McCallum (1923), using a different type of static effort which did not cause such marked fixation of the chest, were unable to confirm Lindhard's results; though the observation of Müller (1939) that the maximum duration of a static effort was not affected by reducing the oxygen content of the atmosphere is in favour of the anaerobic theory. White & Moore (1925) found that during a static effort lasting ten minutes the pulse rate in most cases showed a steady rise, with a rapid fall at the end of the effort. They were chiefly interested in the venous pressure, and concluded that the pumping action of the skeletal muscles was not an essential factor in causing increased venous return during exercise. This question of the absence of the

muscle pump during static effort led to investigation of the effects of this type of effort on the cardiac output, and Grollman (1931) was able to show that active muscular movements caused a much greater increase in cardiac output than static or slow movements. Up to this point the interest in static effort had been mainly metabolic and the recording of the pulse rate was merely incidental. In 1938, however, Asmussen & Hansen attempted to record the changes in respiration, oxygen consumption, minute volume of the heart (by Grollman's method), blood pressure and heart rate in a subject sitting on the floor and extending his legs against moderate or severe resistance. The effort lasted either one minute or three minutes. The pulse rate was recorded from an arm cuff and was used in conjunction with the minute volume to calculate the output per beat. Their pulse rate curves showed a sudden acceleration at the beginning, a plateau maintained during the effort and a very sudden drop in rate immediately after the exercise ended. This rapid post-exercise fall in heart rate was accompanied by a marked increase in the output per beat. They accounted for this by supposing that at the end of the effort the throttling effect of the statically contracted muscle fibres on the circulation disappears, the blood flows rapidly through the dilated muscle vessels back to the heart and shows itself in a secondary increase of the minute volume. In support of the supposed throttling effect

on the circulation they observed that during the static effort there was a marked rise of diastolic blood pressure, which in dynamic work usually only increases slightly. If, as they suggest, the increase in minute volume at the end of the effort is due to a sudden rush of venous blood to the heart it is difficult to account for the simultaneous rapid fall in the heart rate. One would expect that the Bainbridge reflex would either prevent such a fall or would cause a secondary rise in heart rate after a fairly short latent period. No such secondary rise appears in Asmussen & Hansen's figures but it may perhaps be significant that some evidence of such a delayed secondary acceleration occurs in all three of the curves of Fig. 77 following the prolonged static effort. It is possible that two factors, one physical and one mental may combine to produce the very rapid deceleration of the heart rate after prolonged static effort. If the theories of Lindhard and Asmussen & Hansen regarding the throttling effect of the statically contracted muscle fibres are correct, it might be supposed that immediately after cessation of such contraction the muscle capillaries would fill up with blood and thus cause a temporary reduction in the venous return to the heart which would assist the fall in pulse rate. Asmussen & Hansen (1938) actually found that in some cases after severe static effort the minute volume of the

heart did show a sudden brief fall before the marked rise with which they were mainly concerned. In my opinion the mental factor is also important. It is probable that part, at any rate, of the cardiac acceleration during prolonged static effort is due to emotional factors. Such effort is an uncomfortable procedure; the muscles ache, tremor begins and the subject sets his teeth and hangs on. This is bound to have an effect on the heart rate and the sudden removal of the discomfort as the subject relaxes at the end of the effort may play its part in the steep fall of the heart rate.

SUMMARY.

In a brief effort containing both static and dynamic components, it is shown that each of these components has an effect upon the heart rate and that the effects are additive.

The dynamic component causes a brief rapid rise of heart rate, whereas the static component produces a slow steady increase in rate. This is confirmed by experiments on prolonged static effort. The differences of circulatory adjustment to static and dynamic effort are discussed and the findings related to those of other workers.

"As my only wish in presenting these experiments has been to correct a few notions regarding the physiology of the human body, and to advance that estimable science, I shall feel gratified with an examination of my experiments, whether that lead to a refutation or to a confirmation of the opinions maintained throughout this essay".

Robert Knox, (1815), p. 167.

CHAPTER 14.

THE MECHANISM OF CARDIAC ACCELERATION.

It now remains to deal with the various mechanisms which have been described as causing the acceleration of the heart during exercise, and to see how far the results obtained in the foregoing chapters may be interpreted in terms of these. The number of papers on this subject is so great that only the key references are quoted here.

Although there is considerable controversy over points of detail, most workers seem to agree that the following are the factors most likely to play a part in the exercise acceleration of the heart:-

- (a) Influences from the higher centres in the cerebral cortex.
- (b) The blood pressure regulating mechanism, with receptors in the carotid sinus and aortic arch on the output side of the heart.
- (c) The Bainbridge reflex, with receptors in the great veins on the input side of the heart. This mechanism is linked with the question of the output per beat of the heart and with the effect of the pumping action of the muscles during exercise.

(d) Various hormonal, chemical and thermal mechanisms. These are much more indefinite and controversial than the first three.

Most of the above factors operate by sending afferent impulses to the cardiac centres, though some in the last group may affect the pacemaker directly.

The Efferent Nervous Mechanism.

On the efferent side there are the two controlling nerves to the heart, the vagus and the sympathetic. The efferent impulses controlling the heart rate are usually described as originating in the cardio-inhibitory and cardio-accelerator centres in the medulla. The cardio-inhibitory centre seems to be fairly well defined as a result of animal experiments such as those of Miller & Bowman (1915); and, as might be expected, it is closely identified with the vagus nucleus. The existence of a separate cardio-accelerator centre, however, has not yet been proved experimentally; though presumably it would be connected mainly with the sympathetic nervous system. As regards the final distribution of the efferent cardiac nerves there seems to be no doubt that in addition to supplying the sino-auricular node both sets of fibres are also distributed to the conducting system. This was proved for the vagus by the work of Hering (1905b), Erlanger (1909), Robinson (1916),

Kisch (1944) and Jourdan et al. (1945); and for the sympathetic by Hering (1905a), Fredericq (1912), Woolard (1926) and Izquierdo (1929).

It is obvious that the acceleration of the heart rate on exercise might be caused either by an increase of sympathetic tone or by an inhibition of vagus tone or by a mixture of both, and many workers have attempted to define the part played by each during exercise. McWilliam (1893) was of the opinion that the vagus had the major role in the regulation of heart rate and this was confirmed by Hunt (1899), though Hooker (1907) showed that acceleration could take place independently of the cardio-inhibitory centre. All these workers, however, were concerned with the general reflex control of the heart rate rather than with exercise acceleration per se.

The earlier literature, which was very inconclusive, on the nervous control of exercise acceleration was summarized by Gasser & Meek (1914b) who were the first to produce clear experimental evidence. Working on dogs, they showed that at the beginning of voluntary exercise the cardiac acceleration was mainly due to a decrease in vagus tone, though in their view the resting heart rate was largely determined by the sympathetic. This latter observation is contrary to the later work of Crawford (1923) and McDowall (1931) which seemed to indicate that the resting rate was mainly a function of vagal

tone. Samaan (1935a) re-investigated the factors responsible for the exercise acceleration in dogs and confirmed the finding of Gasser & Meek that the immediate acceleration at the onset of work was due to a reduction of vagus tone, though he pointed out that psychical factors play a large part in this event. The persistence of the tachycardia during exercise was attributed to three main factors; continuation of the depressed vagus tone, liberation of adrenaline in response to nervous impulses via the splanchnic nerves; and thirdly, slight augmentation of cardio-accelerator tone. In his view, the chief function of the sympathetic was to provide the necessary 'drive' when the antagonistic vagus inhibition was removed.

Bouckaert & Heymans (1937) obtained similar results but Brouha, Cannon & Dill (1936) found that totally sympathectomized dogs were still capable of acceleration of the heart rate on exercise to a level above that of the denervated heart, and attributed this to the presence of cardio-accelerator fibres in the vagus. Such fibres had previously been described by Morgan & Golland (1932). This phenomenon was further investigated by Brouha, Dill & Nowak (1937) and the contradiction was resolved, in part at any rate, by their observation that a marked exercise acceleration in the absence of the sympathetic does not develop until

about three weeks after sympathectomy. Further experiments by Brouha & Nowak (1939a and b) confirmed this, and the authors again stressed their opinion that the acceleration was due to fibres contained in the vagus and originating in the vagal nucleus. Strong evidence for the existence of such fibres in the dog was presented by Kabat (1940), though in his view they probably play no part in the normal acceleration of the heart due to emotion or exercise. He found that the effects of stimulating these fibres closely resembled those of true sympathetic stimulation; for example, acceleration was produced only after a latent period of several seconds and continued for some time after stimulation had ceased. From this similarity in time relations he concluded that the vagal accelerators were probably adrenergic. Haney, Lindgren & Youmans (1945) also found that such fibres may occur in both vagus nerves in the dog. As was mentioned in Chapter 8, Heymans and his co-workers showed that sinus arrhythmia was mainly due to variations in vagal tone.

The general inference would seem to be that the vagus is by far the more important efferent pathway to the heart, though whether it acts purely through cardiac inhibition or whether it also contains true accelerator fibres in man is very doubtful. In Chapter 10 it was pointed out that certain of the results in the present series of heart

block and auricular fibrillation patients seemed to indicate that when the S-A node was not in control the exercise acceleration of the ventricles showed some of the features characteristic of sympathetic stimulation, though the latent period was such that the possibility of adrenaline secretion could not be excluded.

The Afferent Nervous Mechanism.

The various afferent stimuli which may affect the heart rate will now be discussed in relation to the present results.

(a) Influences from the cerebral cortex.

These would include effects on the resting rate, the anticipatory rise of heart rate immediately before the beginning of exercise and probably the immediate cardiac acceleration at the beginning of the exercise itself. The emotional increase in heart rate which occurs shortly before exercise begins was discussed at some length in Chapter 6, when it was shown to appear very markedly in the normal subjects. In the experiments on the effect of load (Chapter 9) it was found that this anticipatory increase in heart rate was especially prominent and was approximately a function of the load to be carried, even though the effects of the load were not felt in any way until exercise actually commenced. This was confirmed in the S.T.C. Series (Chapter 11).

In contrast to the normal subjects, the psychological increase prior to exercise was found to be either absent or much diminished in the cardiac patients of all groups. In some cases this was no doubt due to the fact that the resting rate was already much above the normal level.

The actual mechanism by which emotion alters the heart rate remains obscure, though a good deal of attention has been paid to it. There are really two problems here; the first being that of the anticipatory increase prior to exercise, and the second the question of whether the higher centres play any part in the immediate acceleration at the beginning of exercise itself. The first question would seem to be allied to the changes of heart rate which occur during fear, anger and other general emotions. Gillespie (1924) showed that mental work could produce a considerable increase in heart rate but his experiments gave no indication of the mechanism involved. Britton, Hinson & Hall (1930) analysed the factors involved in the regulation of the heart rate of the cat during emotional excitement and found that the vagus, the sympathetic and the secretion of adrenaline each played a part. The observation of Fleisch (1933) that the pulse rate of psychical excitement could be lowered by increasing vagal inhibition through the oculo-cardiac or Valsalva reflexes does little to solve the problem, as it is generally agreed

that strong vagal inhibition takes precedence over sympathetic stimulation (Kuntz 1929). The rapidity with which emotional changes may be reflected by the heart rate led Whitehorn, Kaufman & Thomas (1935) to suppose that they must be mediated through release of vagal inhibition. Contrary results were, however, obtained by Bond (1943) who concluded that the sympathetic accelerators were largely responsible. It also seems clear from the recent work on effort syndrome that psychological influences can maintain an elevation of the resting heart rate over very long periods of time.

Whatever their final path, it is to be presumed that emotional impulses have their origin in the higher centres, the most likely situations being the cortex or the hypothalamus. In this connexion it is of interest that Hoff & Green (1936) obtained cardiac acceleration by stimulating various areas of the cortex in cats and monkeys. That such paths must exist is also shown by the sporadic cases of true voluntary acceleration of the heart rate which occur throughout the literature. One such case was carefully studied by Favill & White (1917) who produced evidence that the voluntary increase was chiefly due to stimulation via the sympathetic, and not to withdrawal of vagal inhibition. A similar case was investigated by Carpenter, Hoskins & Hitchcock (1934) and this subject was found to be capable of voluntarily increasing his heart rate

by twenty five per cent. over the initial level.

As regards the second question, whether impulses from the higher centres play a part in the immediate acceleration of the heart rate at the beginning of exercise, several workers have shown that the latent period for this acceleration may be less than one cardiac cycle and this is confirmed by the present tracings (see Chapter 6). This fact certainly indicates a nervous mechanism. Johansson (1895) advanced the theory that impulses along the motor paths to the voluntary muscles may affect the cardio-accelerator centre in the medulla, though he gave no reason for choosing the accelerator centre rather than the inhibitory one. Aulo (1909) and Martin & Gruber (1913) supported Johansson's theory as a result of experiments in which passive muscular movements caused no cardiac acceleration, in marked contrast to even mild voluntary movements. Unlike Johansson, they considered that the cardio-inhibitory centre was depressed as a result of impulses from the motor cortex. A similar theory was also put forward by Krogh & Lindhard (1913). An observation made by Samaan (1935a) may be significant in this connexion. Dogs trained to exercise on a treadmill were placed on a stationary board above the apparatus, and it was found that immediately the treadmill beneath was started there was an acceleration of the

heart which lasted for five to ten seconds, although the dogs had made no movement. This, presumably, might raise the question of how far such an acceleration is in the nature of a conditioned reflex.

The results of Jacobson (1929 & 1930) may throw some light on the mechanism of both the 'anticipatory' and the 'exercise' psychological accelerations. He found that if a subject imagines or even recollects various muscular movements, action potentials appear in the muscles in question. Thus the 'idea' of an act may cause a state of 'subminimal' activity in the organ concerned so that it is the more prepared for the actual performance of the act, a theory which may well apply in the case of the heart.

From the evidence given above, it would appear that the anticipatory acceleration may be brought about mainly by sympathetic stimulation, whereas the immediate acceleration on exercise is almost certainly due to release of vagal inhibition. If psychological influences play a considerable part in the immediate acceleration, as they probably do, it seems curious that the final mechanism should be different in the two cases. There seems to be no doubt, however, that the time relations of the two accelerations are different; the anticipatory acceleration appearing gradually whereas the exercise acceleration commences abruptly.

(b) Reflexes from the working muscles and from the output side of the heart.

In 1898 Athanasiu & Carvallo put forward the theory that the working muscles send afferent impulses to the central nervous system which depress the cardio-inhibitory centre. Their evidence was very inconclusive, but recently Alam & Smirk (1938) have described a reflex mechanism in man by which metabolites accumulating in active muscles cause acceleration of the heart rate. The time-relations, however, make it most unlikely that this reflex takes part in the initial acceleration of exercise, though the authors consider that it may play a minor role in the acceleration during vigorous exercise. Asmussen, Nielsen & Wieth-Pedersen (1943) performed experiments in which muscles were caused to contract by electrical stimulation and concluded that when the 'steady state' is reached the circulation is controlled by reflexes from the working muscles rather than by impulses from the cerebral cortex.

It has long been known that a rise in blood pressure causes a fall in heart rate and vice versa, and this mechanism has been worked out in detail, chiefly by Hering, Starling and Heymans and their collaborators in the investigation of the sino-aortic reflex (see Anrep, 1936). A simple method of demonstrating this reflex in man was given by Asmussen, Christensen & Nielsen (1938). By compressing and relaxing

the bloodflow through both legs they caused marked alterations in the blood pressure, and the pulse rate clearly showed the inverse changes. As a result of experiments on the effects of gravity on the human circulation, Asmussen & Knudsen (1942) concluded that the pressure-sensitive reflex which controls the blood pressure does not control the cardiac output. A somewhat similar view had previously been suggested by Hess (1930).

It is indeed obvious that during muscular exercise, when the heart rate and blood pressure rise together, the sino-aortic reflex mechanism is largely overruled. It would seem that this afferent pathway produces its main effects on the vasomotor centre and that Marey's law is based on a relatively weak reflex arc. Some evidence that the carotid sinus reflex may actually assist the rise of blood pressure during exercise has been put forward by McDowall (1941).

(c) Reflexes from the input side of the heart; the output per beat.

A great deal of work has been done on the right auricular reflex since it was first described by Bainbridge (1915). An account of some of these investigations has already been given in the discussion on auricular fibrillation (Chapter 10). As a result of animal experiments Sassa & Miyazaki (1920) concluded that the Bainbridge reflex operates chiefly through diminution in vagus tone but also partly

through augmentation of accelerator tone. It is extremely difficult to assess the importance of the Bainbridge reflex during a brief exercise in the human subject. The results of McCrea, Eyster & Meek (1928) seemed to indicate that during prolonged exercise there was a definite balance between the venous pressure and the heart rate, and that the more severe the exercise the more consistent were the results. In such exercise the venous pressure may rise by about 100 mm. of water and this was thought to be sufficient to trigger the right auricular reflex in man. Sharpey-Schafer & Wallace (1942) found that when large quantities (up to 2000 c.c.) of saline, serum or blood were rapidly injected into the veins of normal subjects there was frequently no increase of heart rate. Examination of their experimental data shows, however, that if the venous pressure increased by over 90 mm. of water there was usually an increase in heart rate. It is thus possible that even in severe exercise the threshold value for the right auricular reflex in man is scarcely exceeded. This gives support to the earlier view of White (1924) that this reflex cannot be so important during exercise as is usually believed. This does not imply, of course, that the activity of the muscle pumps in keeping the heart well supplied with blood during exercise is any less important.

Another factor which is clearly linked with the increase in heart rate during exercise is the question of the

output per beat of the heart. As is well known, there are two main possibilities concerning the behaviour of the heart when an increased demand is made on the circulation. The first theory postulates that the stroke volume is variable, and that the reserve capacity of the circulation lies in variations of both the output per beat and the frequency of the heart. The second theory postulates that the output per beat remains nearly constant, and that the demands on the circulation during activity are met mainly by variations in the frequency of the heart beat. A factor of safety is provided during exercise because the tissues are then able to take more oxygen out of a given volume of blood (increased coefficient of oxygen utilisation). This theory of the constant output per beat was supported by Henderson (1923 & 1925), and Henderson, Haggard & Dolley (1927) found that the output per beat remained relatively constant during rest and moderate exertion in ordinary subjects, but that it might be considerably increased in athletes performing strenuous exercise.

The alternative theory, that of variable output per beat, was put forward by Bainbridge (1919) and was supported by the experiments on man carried out by Means & Newburgh (1915), White (1924), Lythgoe & Pereira (1925) and Liljestrand, Lysholm & Nylin (1938). Animal experiments by Tappan & Torrey

(1926), Marshall (1926b) and Harrison, Blalock, Pilcher & Wilson (1927) also confirmed this theory, which is now held by most workers. An interesting corollary to the theory of variable output per beat was stated by Plesch (1937).

In his view, when the minute-volume of the heart is increased, the rate and output per beat are regulated so as to produce minimum strain on the heart muscle. Starling & Visscher (1926) observed that, within limits, slowing the heart enables it to do a given amount of work more economically. On Plesch's theory one might expect that after exercise has begun and the venous return has increased, the greater output per beat would permit a diminution in the cardiac acceleration. This was at first thought to be a possible explanation of the decrease in heart rate observed towards the end of exercise in Series A and B (Figs. 13 and 14), but this was later proved to be due to the withdrawal of the postural component of the exercise.

As was mentioned above, it is known that athletes' hearts are usually capable of considerable increase in the stroke volume during effort, and this no doubt partly accounts for the relatively low maximum heart rate observed in an athlete during a six mile run (Chapter 12). The results of Tuttle & Salit (1945) are in agreement with this.

The general statement of Harris & Lipkin (1931) that: "the normal individual reacts to exercise as far as

possible by increase in stroke-volume, the diseased more by an increase in the pulse frequency", is difficult to reconcile with the relatively small increase in heart rate on exercise shown by many of the present cardiac patients, though it may hold for the special case of auricular fibrillation. This problem was fully discussed in Chapter 10.

(d) Chemical and Physical influences affecting the heart rate.

A large number of attempts have been made to determine whether the adrenals play any part in the acceleration of the heart rate during exercise. Gasser & Meek (1914a) as a result of experiments on dogs concluded that the secretion of adrenaline may be a factor of importance during severe exercise, though later (1914b) they attributed the adrenaline secretion to the asphyxia caused by vagotomy. The classical investigations of Cannon & Britton (1927) on the cat seemed to indicate, however, that comparatively mild exercise, such as walking, could cause an increase in heart rate through adrenal secretion. Bond (1943) showed that in cats and dogs emotion may also affect the heart rate by causing secretion of adrenaline, but only after a latent period of about twelve seconds. Pilcher, Wilson & Harrison (1927) found that adrenaline increased the cardiac output of normal dogs. Terry & Peters (1933) observed that adrenaline can cause ventricular extrasystoles, and it is

possible that adrenaline secretion may have played a part in causing the delayed extrasystoles seen after severe exercise in one of my heart-block patients (Fig. 39). It is, however, extremely difficult to differentiate the possible effects of direct sympathetic stimulation of the heart in man from those which might be brought about via stimulation of the adrenals.

With regard to the effect of muscle metabolites on the heart rate, Petersen & Gasser (1914) were of the opinion that these substances have no significant part in the exercise acceleration; but Cannon, Linton & Linton (1924) suggested that though they have no direct effect on the heart rate they may produce acceleration by stimulating the adrenals.

It has been shown that a rise in the carbon dioxide content of the blood may accelerate the heart (Schneider & Truesdell, 1922b; Marshall, 1926a and McDowall 1929) but the concentrations required are such as to render it unlikely that this effect is important during normal exercise.

The effect of increased temperature of the blood during exercise was at one time considered to be of importance, particularly in causing the persistent increase in heart rate during the post-exercise period (Mansfeld , 1910). Later work by Gasser & Meek (1914a) on animals, and by Martin, Gruber & Lanman (1914) on man, showed that the temperature factor is

probably of negligible significance except during very prolonged and severe effort.

The Interrelations of the Various Factors.

Hunt (1897) attempted to determine the effect on the heart rate of simultaneously stimulating the vagus and accelerator nerves at various frequencies. Working on dogs, cats and rabbits, he concluded that the result of simultaneous stimulation of the two nerves was approximately the algebraic sum of the results of stimulating them separately. In 1934 Rosenblueth & Simeone performed similar experiments on cats and obtained a different result. They found that the effect of simultaneous stimulation of the two sets of nerves was not the algebraic sum but the resultant of the two influences. In their view, stimulation of the accelerators or decelerators multiplies the existing rate by a factor which may be greater or less than one, and does not simply add or subtract a certain number of beats. When simultaneous stimulation occurs the two multiplicative effects take place independently, as if each set of nerves was acting alone. It is very difficult, however, to say how far the results of these animal experiments can be applied to the delicate control of the heart rate in man. Darrow (1942) was of the opinion that the clinical value of the pulse rate depended little or not at all on its role as a sympathetic -

parasympathetic indicator.

Conclusions from the Present Experiments.

The heart rate during the step test is probably the resultant of three main factors; psychological influences, posture and the actual stepping exercise. When an attempt was made (Chapter 7) to analyse the effects of the postural and exercise components, the emotional factor was kept as constant as possible by using subjects who were well accustomed to the apparatus. One of the remaining factors was then eliminated while all the other conditions of the experiment remained as before. The same methods were employed in the analysis of the effects of the 'static' and 'dynamic' components of the spring-pulling test (Chapter 13).

Under these circumstances it was possible to show that even relatively minor components of a mixed exercise cause a definite effect on the total heart rate curve during work. Too little is known of the exact pathways by which such small increments of work produce their effects on the heart rate to permit of any statement as to whether the responses must be mediated through similar channels in order that they may be additive. As regards the additive effects of the dynamic 'postural' component and the 'exercise' component at the beginning of the step test, it might reasonably be supposed that these are mediated by the same

pathways, since the type of movement is the same in both. This may not be the case, however, with the 'static' and 'dynamic' components of the spring-pulling test.

The time-relations of the cardiac accelerations due to the various components are also of interest. For example, in the case of the dynamic postural component of the step test the act of standing erect only occupies about two seconds, but the resulting cardiac acceleration does not reach its maximum until after about eight seconds and the heart rate does not settle to the level dictated by the gravity component until after about twenty seconds (Fig. 15, Curve C). In the mixed exercise these time-relations are strictly adhered to, as was well shown in the duration experiments (Fig. 22). It is as if each component gave rise to a burst of activity which lasted for a definite time and then declined, and this rise and decline seems to produce its effect on the heart rate even though other components with different time-relations are acting simultaneously. This simple relationship only appears to hold, however, if the simultaneous components are reasonably similar in strength. If one component is much more powerful than the other, the effect of the weaker is no longer detectable. This was demonstrated in Fig. 20, in which the effect of the decline in the postural component of the step test disappears when the

exercise component increases with heavier loads. Similarly, though the rise and fall of heart rate due to the dynamic component is clearly visible when pulling out the springs at the beginning of the prolonged static effort test, when the static component is relatively small; the 'release' acceleration at the end of the prolonged effort is not detectable, as the static component is then exerting such a powerful influence (Fig. 77). This release acceleration is easily seen, however, at the end of a brief static effort (Fig. 72). The fact that a small addition to the work is much more effective in producing an increase in heart rate at the beginning of exercise when the heart rate is low than later when it is high, would favour Hunt's additive theory rather than Rosenblueth & Simeone's multiplicative one.

A similar rule seems to hold for the total effect of exercise on the heart rate, as it was shown in Chapter 6 that where the initial rate was high the absolute increase in rate produced by a given exercise was generally small. Extreme examples of this were also given in Chapter 10 in the section on hyperthyroidism. The matter was discussed on p.59 in relation to the theory of Cotton, Rapport & Lewis (1917a) that "the circulation is capable at a given moment of a certain response to a given effort, and it appears to be a matter of indifference whether this response has been called

forth to some extent before the chief stimulation has been applied providing it has been called forth through similar channels". A corollary to this would be that the maximum rate of the heart during exercise is governed by the metabolic needs of the body rather than by the actual signals of the intensity of the exercise, which presumably would tend to raise the rate by a more or less standard amount for a given exercise.

The present results also afford some support to the general theory that the initial acceleration during exercise in man is due to a sudden reduction in vagal tone, but that later in the exercise the effects of sympathetic stimulation, and possibly of adrenaline, make their appearance (Bowen, 1904; Samaan 1935a). As was pointed out above, the earlier work on the very brief interval between the commencement of exercise and the onset of cardiac acceleration is fully confirmed by the present tracings in normal subjects, and the time-relations are such that only withdrawal of vagal inhibition would account for them. Evidence has also been given in Chapter 10 that this immediate acceleration is very much reduced in cases of auricular fibrillation and heart block, so that control through the normal pacemaker would seem to be essential for its occurrence. It is impossible to say from the present results whether this initial acceleration

is purely psychological or whether the emotional increase is reinforced by other means such as impulses from the working muscles. It may be significant that in those conditions in which the immediate acceleration is subnormal, the pre-exercise psychological increase in heart rate is also very much diminished or even absent. A further point is that the immediate acceleration (during the first five seconds of exercise) varies very little with the load carried (Fig. 20), though later the curves diverge according to the load. A similar effect is seen in the patients with valvular disease (Fig. 26) in whom the differing valvular disabilities would act as varying loads. These results would seem to indicate that whatever the actual mechanism which signals the work load, it does not become effective until after the first few seconds of exercise. This is not the case with the effect of rate of working, as Fig. 24 shows that the acceleration during the first five seconds of exercise is less at 48 steps per minute than at 96 per minute. It may be significant that work load is a condition imposed from outside, so to speak; whereas rate of stepping is presumably dictated from the cerebral cortex. In general, it was shown in Chapter 9 that the rate of working chiefly affected the increase of heart rate during the exercise, whereas the duration of work largely determined the time of return to normal. This might be

explained by the fact that a longer duration of exercise would allow the secondary factors such as the sympathetic and adrenaline to exert their influence, as these are known to have a much longer latent period and a longer 'afterdischarge' than effects mediated through the vagus.

In 1946 Gray put forward quantitative evidence for a multiple factor theory of the control of respiratory ventilation, i.e. that the amount of lung ventilation is determined by the sum of the partial effects of the chemical, thermal and reflex agents affecting respiration. It would appear likely that a somewhat similar mechanism is responsible for the control of the heart rate.

One of the main points brought out by this work is that relatively minor components of the total heart rate have a detectable effect on the total heart rate. The bearing of this on the interpretation of the various heart rate components during exercise is discussed in the following section.

SUMMARY.

The various mechanisms, nervous and otherwise, which may bring about the acceleration of the heart rate during exercise are described and key references given.

The anticipatory rise in heart rate before exercise begins is discussed in detail and it is shown that the present results favour the theory that the immediate acceleration at the beginning of exercise is also largely psychological. A condition imposed from outside, such as work-load, does not affect the immediate acceleration, though it may cause a marked anticipatory rise of heart rate.

With reference to the interrelations between the various factors it is shown that the present results favour an additive theory rather than a multiplicative one.

One of the main points brought out in the present study is that relatively minor components of a mixed exercise produce a detectable effect on the total heart rate curve during work. The bearing of this on the interrelations of the various heart rate components during exercise is discussed.

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The Effect of Exercise on the Heart Rate

by

J.A.C. Knox, M.B., Ch.B.

Volume 2.

(Figures and Tables)

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Figures 1 to 77

followed by

Tables 1 to 13

NOTE

With the exception of Figs. 6 and 8 all the figures were reproduced by the Kodaline process. The graphs were reduced photographically from ink drawings. The reproductions of actual smoked drum tracings were made by printing directly by contact on to Kodaline paper, so that these tracings are reproduced natural size. No re-touching was performed, and therefore any slight creases or blemishes on the original will also appear on the Kodaline print.

Fig. 1. Reprinted at p. 17 from Richard's "New
piano" 2nd edition, 1700.

likewise for its importance, deserves all our attention. What I mean is, the frequency or quickness of the pulse, which, though distinguished by some writers, I shall use as synonymous terms. This is generally the same in all parts of the body, and cannot be affected by the constitutional firmness or flaccidity, or smallness or largeness of the artery, or by its lying deeper or more superficially; and is capable of being numbered, and consequently of being most perfectly described and communicated to others.

THE degrees of quickness of the pulse, belonging to the several ages and distempers, have been taken notice of by few physicians in their writings; and, as many observations are necessary to settle this doctrine, what I have made, and am going to relate, may be of use towards confirming, correcting or enlarging those, which

Fig. 1. Facsimile of p. 20 from Heberden's "Remarks on the pulse" 2nd Edition, 1786.

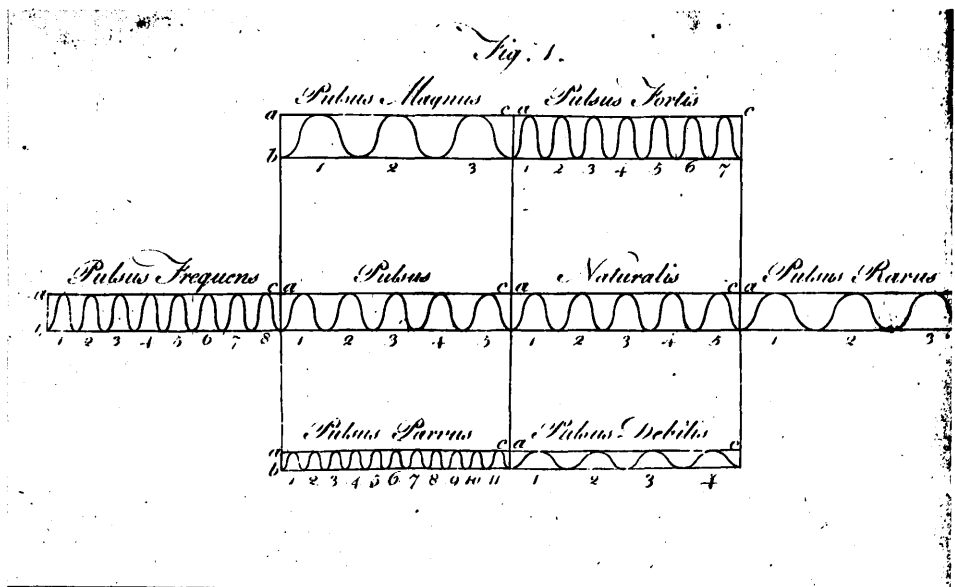


Fig. 2. Facsimile of plate I from Stedman's "Physiological essays and observations", 1769.

dentary life, in both cases the pulse ceases to be equal, flexible, elevated, developed, and free, such as is observed in adults enjoying the best health, who do not neglect corporeal exercise: on the contrary, in the excessive exercise of the body, it becomes rapid, tempestuous, violent and irregular, as may be seen in the case of grotesque dancers, runners, blacksmiths, and all such as are engaged in violent or fatiguing labours. In the course of such bodily exercises and labours, the fact is always to be observed, that the columns of blood crowd precipitately into the ventricles of the heart, which, in order to disembarass themselves of this accumulation of blood, are compelled to repeat their contractions with like frequency; and they repeat them with a sensible impetus, as if they were irritated, so to speak, with the quick return of the blood, which incommodes them; and from this state of violence of the ventricles of the heart, and vascular sanguineous system, is derived the rapidity, agitation, vehemence and disorder of the beatings of the pulse, which characterize the irregular and violent exercises of the body. Sometimes those exercises of the body are so violent and strong, and consequently the course of the blood so

Fig. 3. Facsimile of p. 222 from Rucco's "Introduction to the science of the pulse". Vol. 1, 1827.

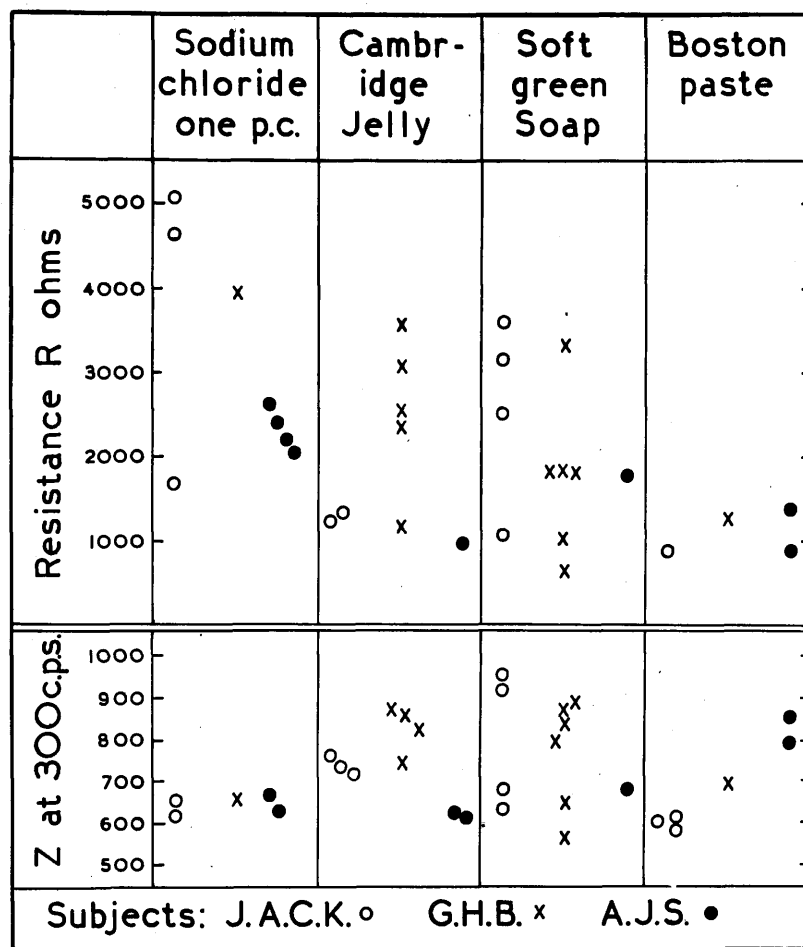


Fig. 4. Chart of all the observations of body resistance and impedance from forearm to forearm for the three subjects with the four different electrolytes.

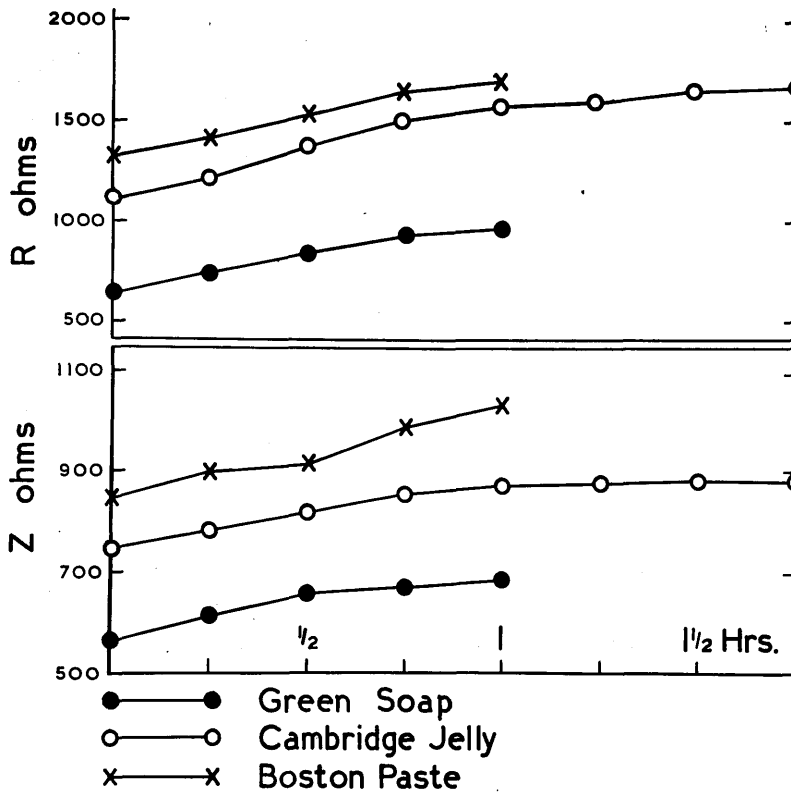


Fig. 5. Variation of resistance (forearm to forearm) with time.

Cambridge jelly and Green soap, subject G.H.B.

Boston paste, subject A.J.S.



Fig. 6. General view of recording apparatus.

The leads from the subject can be seen descending from the overhead trapeze (not shown). The amplifier and writing mechanism are visible in the background.

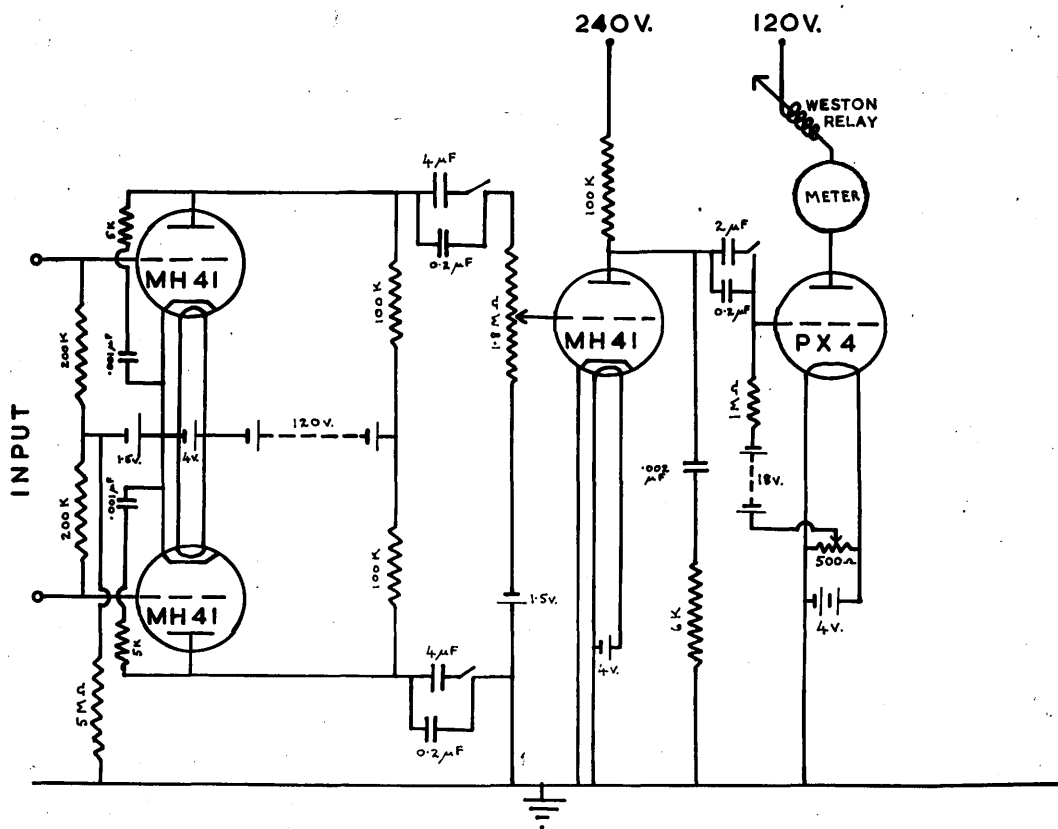


Fig. 7. Circuit diagram of amplifier used for recording the cardiac action potentials.

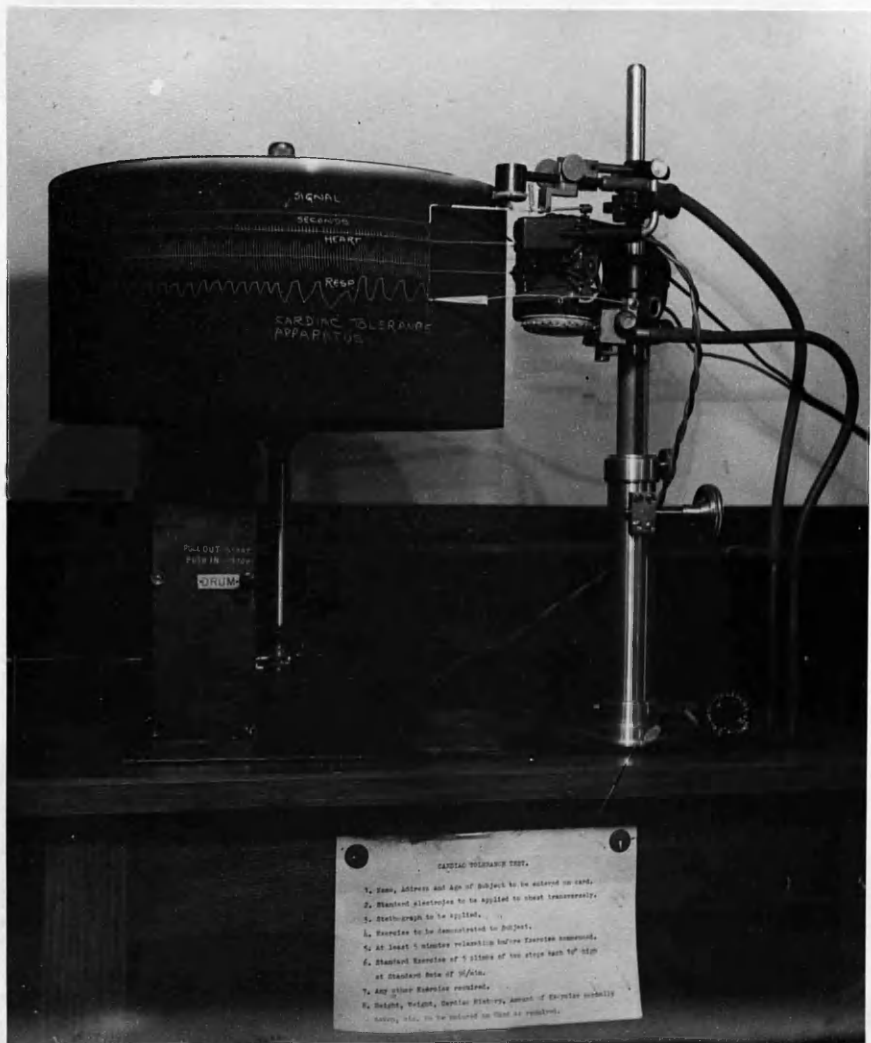


Fig. 8. Writing mechanism and drum.

The pointers, from above downward, record:

Signal	- beginning and end of exercise.
Time	- in seconds.
Heart Beats	- R-S waves of electrocardiogram.
Respiration	- a downward movement indicates inspiration.

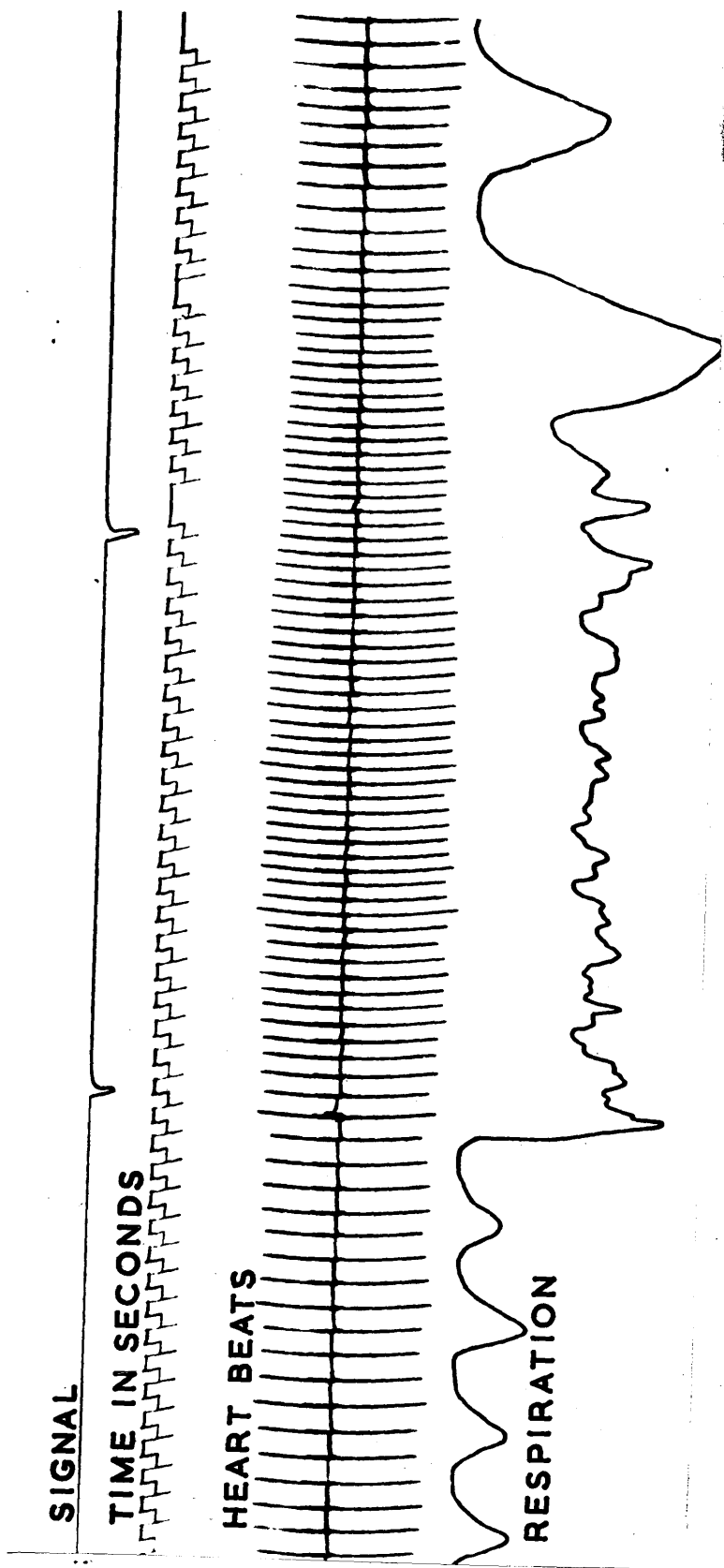


Fig. 9. Example of a typical tracing.

Fig. 10. Kymograph tracing with vertical lines for analysis of the heart rate changes during exercise.

The lower portion of the tracing is continuous with the upper portion.

For an explanation of the lettering, see Chapter 4.

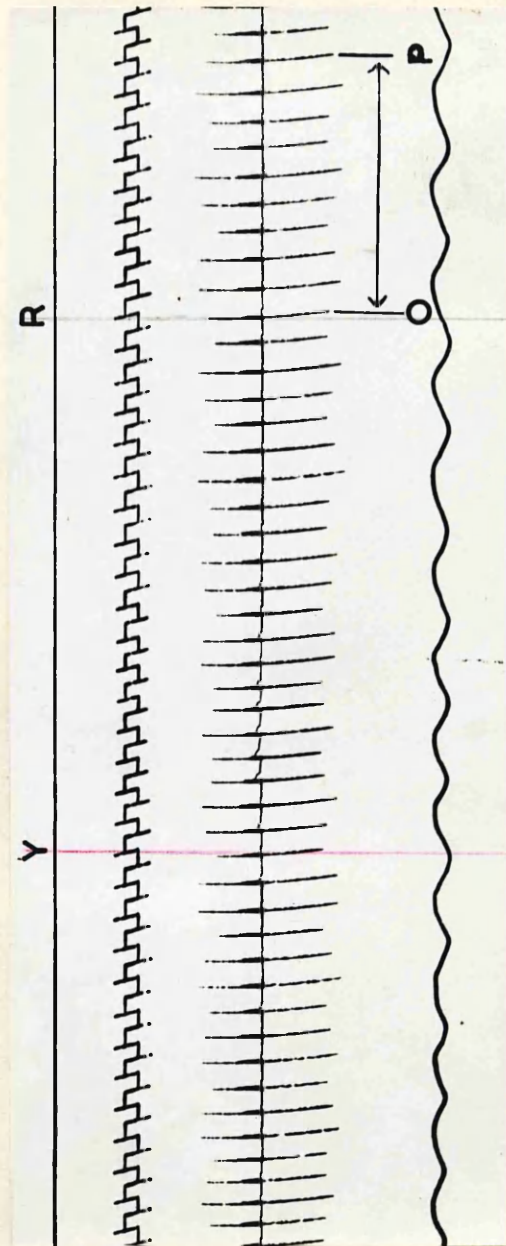
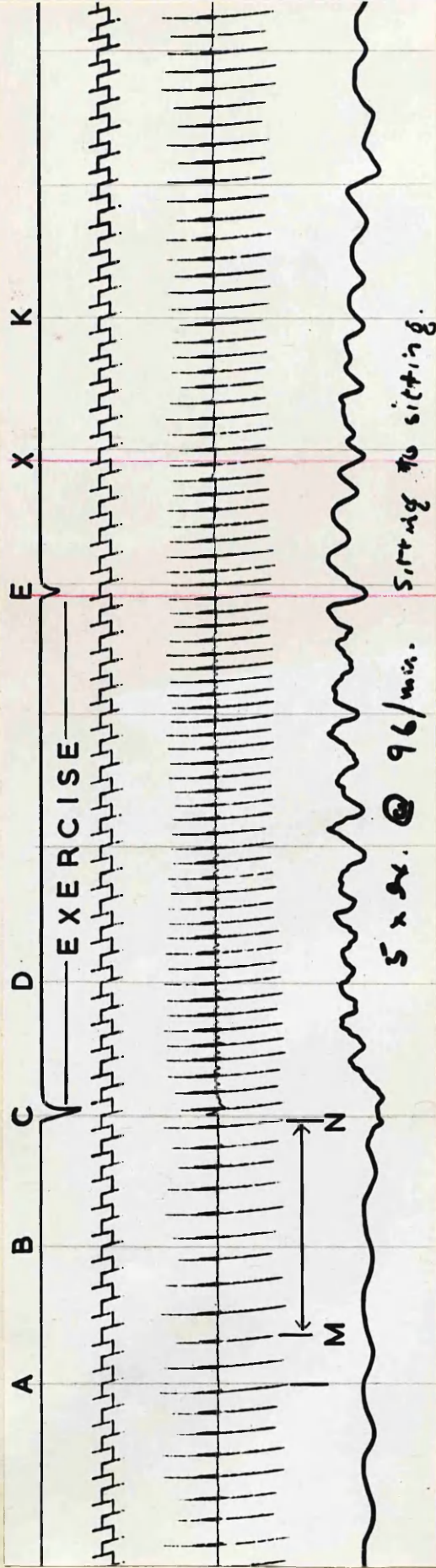


Fig. 10.

Name James Smith.

Trace No. A 24

Address 27 Caird Drive Glasgow W1.

Age 21 M ~~or F~~ Ht. 5' 9.9" Wt. 65.6 Kg. Leg 3' 1"

Chest: In $34\frac{1}{4}$ Out $31\frac{1}{2}$ ($2\frac{3}{4}$). Hb 83 % B.P. $\frac{118}{67}$
(Dave)

Cardiac history:

No relevant history.
No rheumatic fever, scarlet fever etc.
Takes only moderate exercise, occas. walks, golf.

Fig. 11. Example of standard record card.

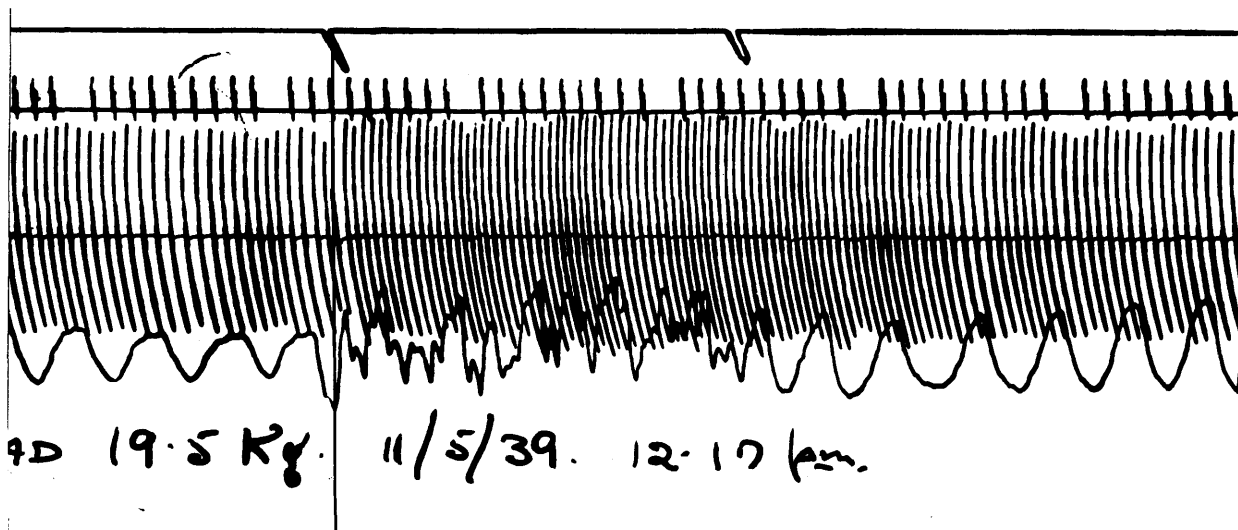


Fig. 12. Kymograph tracing showing the very brief latent period between the beginning of exercise and the onset of cardiac acceleration.

Vertical ink line indicates the beginning of exercise.

Upper line - signal
 Second line - time in seconds
 Third line - heart beats
 Fourth line - respiration.

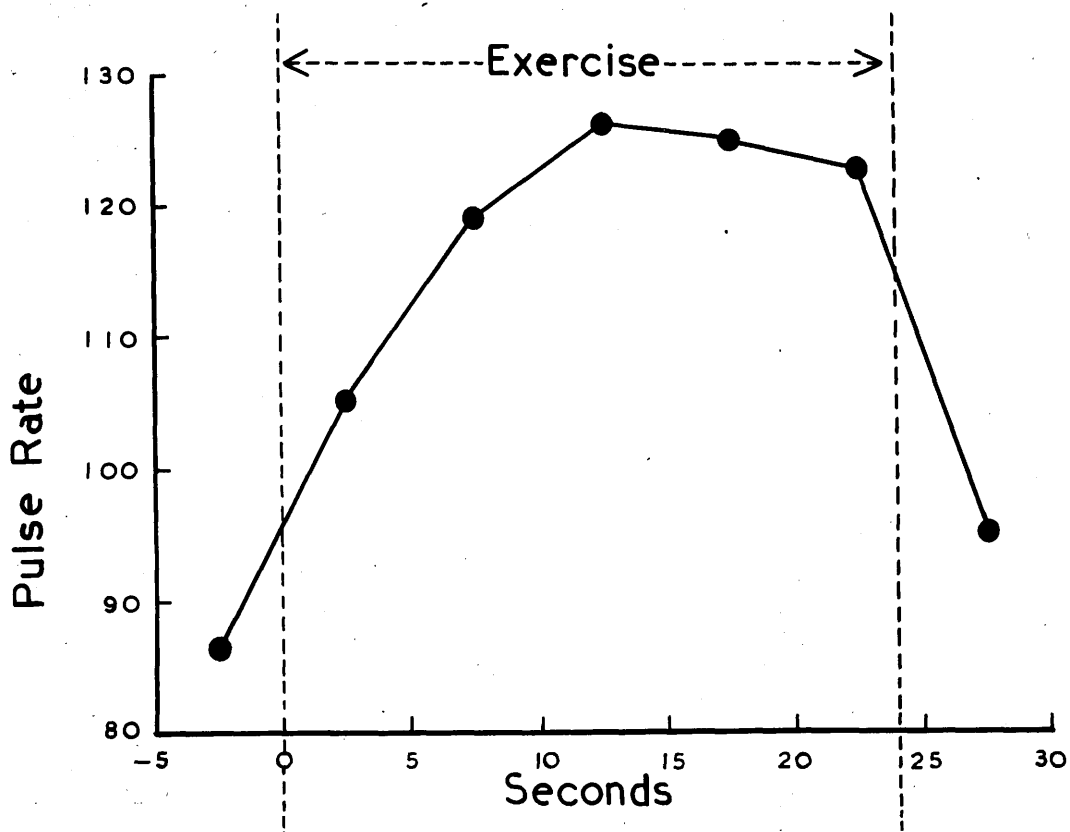


Fig. 13. Graph of average heart rates in 5 second intervals before, during and after the standard exercise. Series A, 75 male students.

The average maximum rate here is slightly lower than that given in Table 1 because the maximum rate does not always fall in the same 5 second interval.

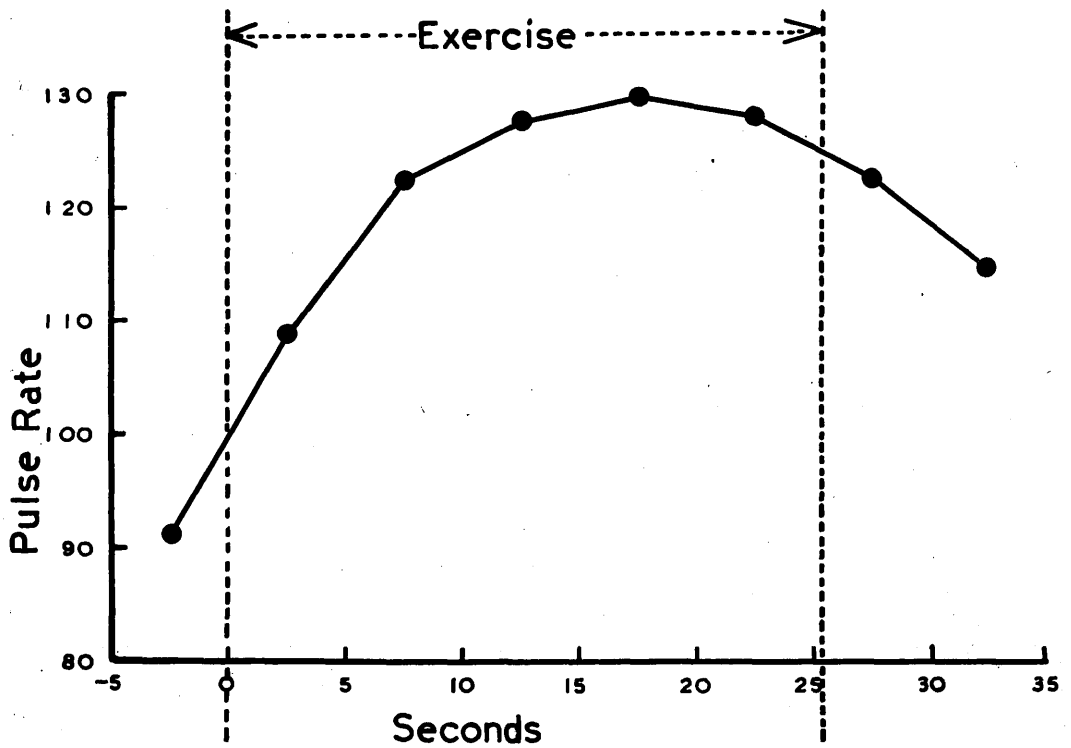


Fig. 14. Graph of average heart rates in 5 second intervals before, during and after the standard exercise. Series B, 25 female students.

EFFECT OF POSTURE DURING THE STANDARD EXERCISE.

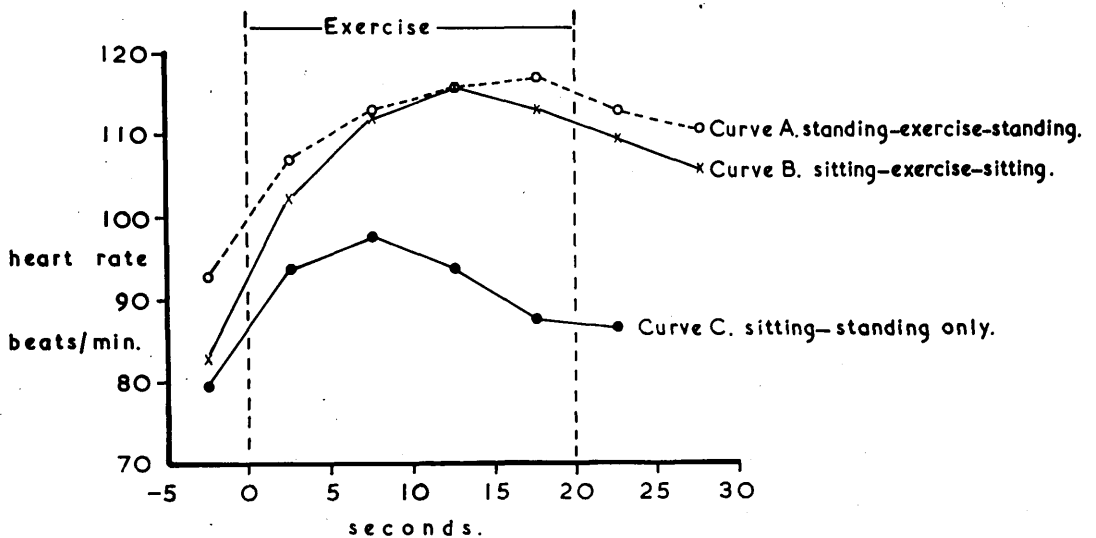


Fig. 15. Graphs of mean heart rates in 5 second periods during the standard exercise, and the effect of posture alone. (7 male subjects)

Curve A - exercise performed beginning and ending in the standing posture.

Curve B - exercise performed beginning and ending in the sitting posture.

Curve C - mean heart rates when the subjects changed from the sitting to the standing posture.

EXERCISE COMPONENT OF HEART RATE.

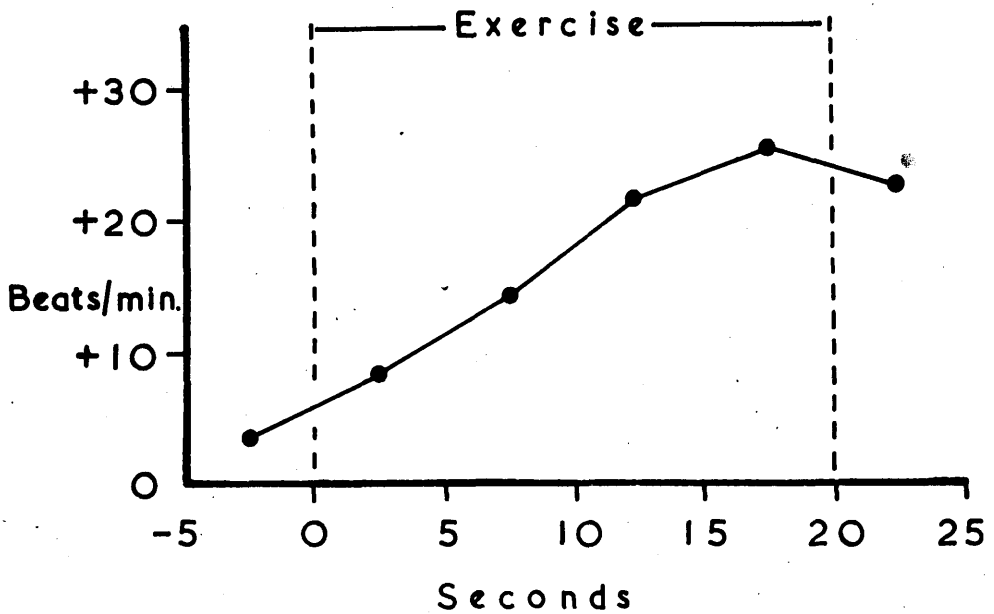
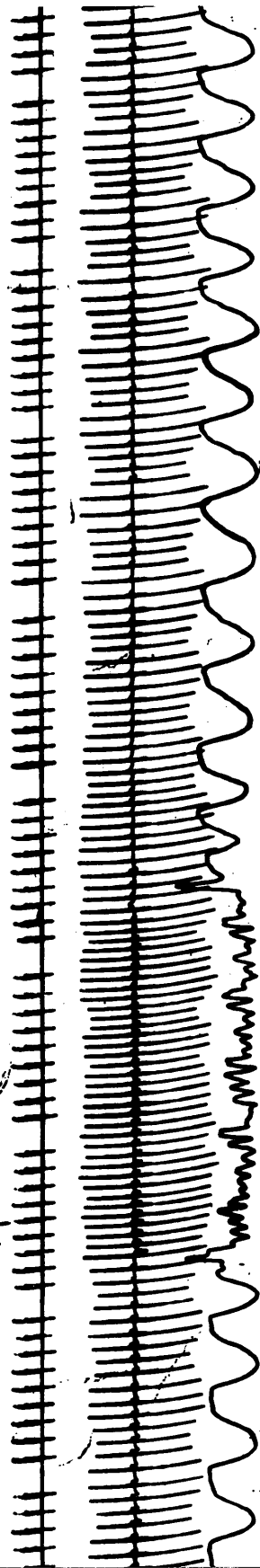


Fig. 16. Graph of heart rate in successive 5 second periods obtained by subtracting curve C from Curve B (Fig. 15). This gives the rise of heart rate due to the exercise component alone. (7 male subjects)

57—EXERCISE—



27/5/29. 11.23 AM NO LOADS 570X @ 96h.

Fig. 17. Kymograph tracing showing marked sinus arrhythmia.

The respiratory arrhythmia is present before exercise, disappears during the exercise and is most marked shortly after exercise ends.

A downstroke on the respiratory tracing indicates inspiration.

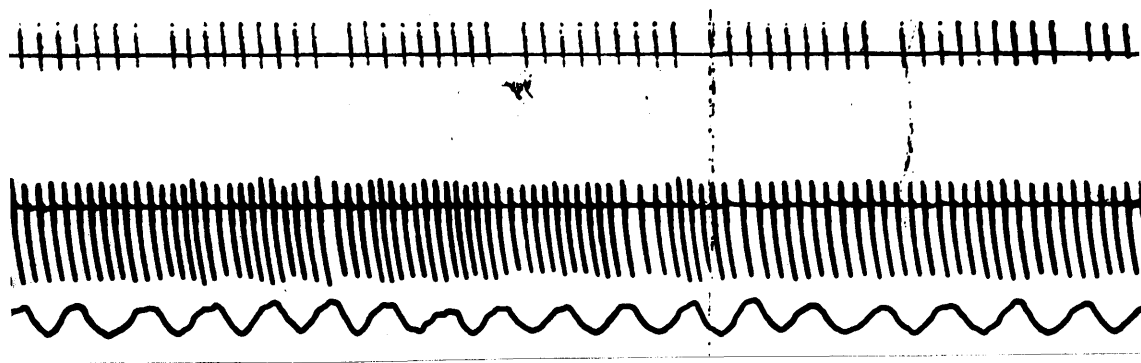


Fig. 18. Kymograph tracing taken shortly after the end of exercise and showing simple pauses in the rhythm of the heart.

Upper line -- time in seconds
Middle line - heart beats
Lower line -- respiration

EFFECT OF LOAD ON VARIOUS H.R. INDICES

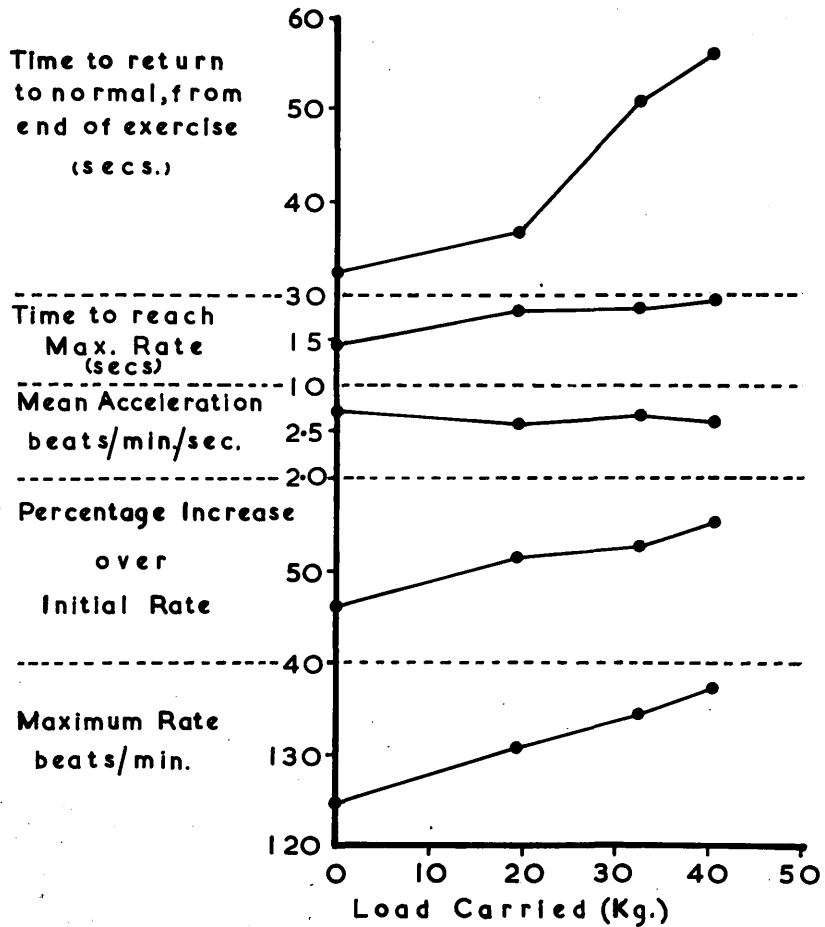


Fig. 19. Series W. Graphs showing the effect of load carrying on various heart rate indices. Standard exercise of 5 ascents at 96 steps per minute.

EFFECT OF CARRIAGE OF A LOAD VARIOUS WEIGHTS

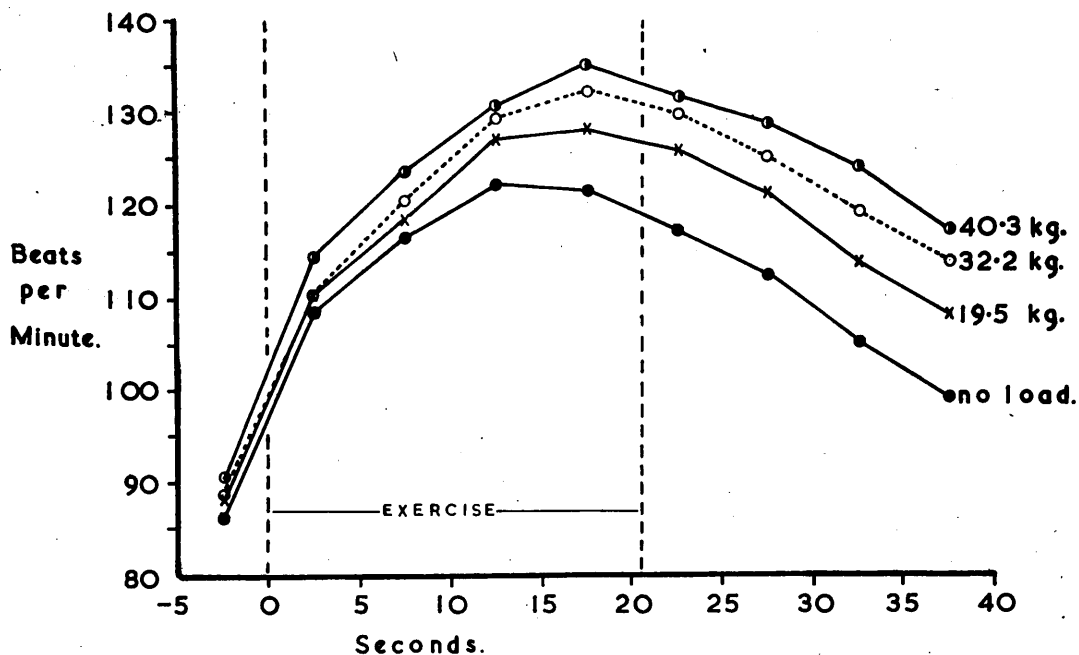


Fig. 20. Series W. Graphs of mean heart rates in 5 second intervals during the standard exercise, showing the effect of carrying various loads.

EFFECT OF DURATION OF EX. ON VARIOUS INDICES

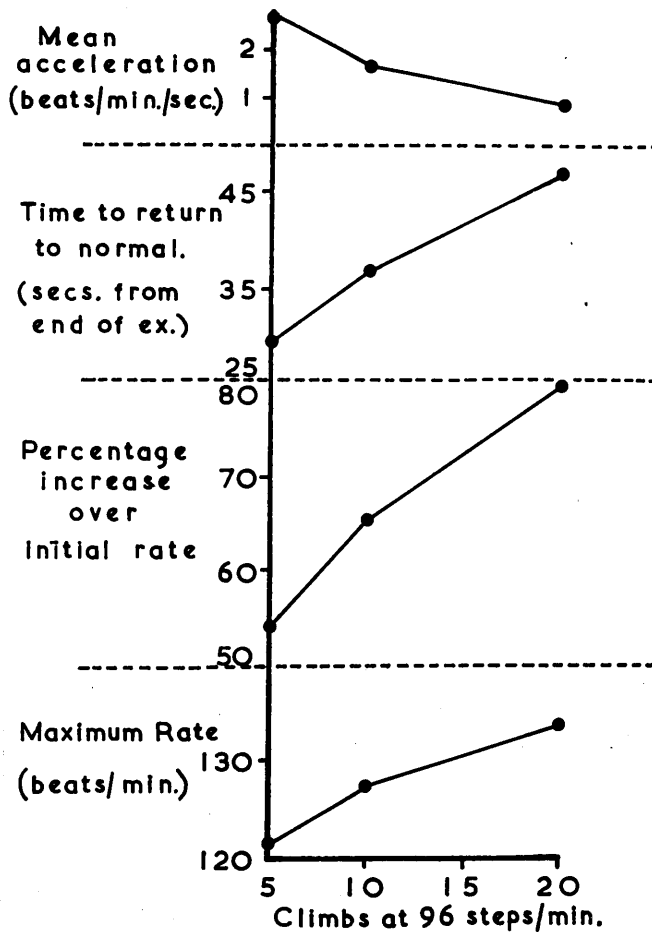


Fig. 21. Series D. Graphs showing effect of duration of exercise on various heart rate indices.

The exercise consisted of 5, 10 and 20 ascents of the two steps at the standard speed of 96 per minute.

5, 10 & 20 climbs at 96 per minute. No Load.

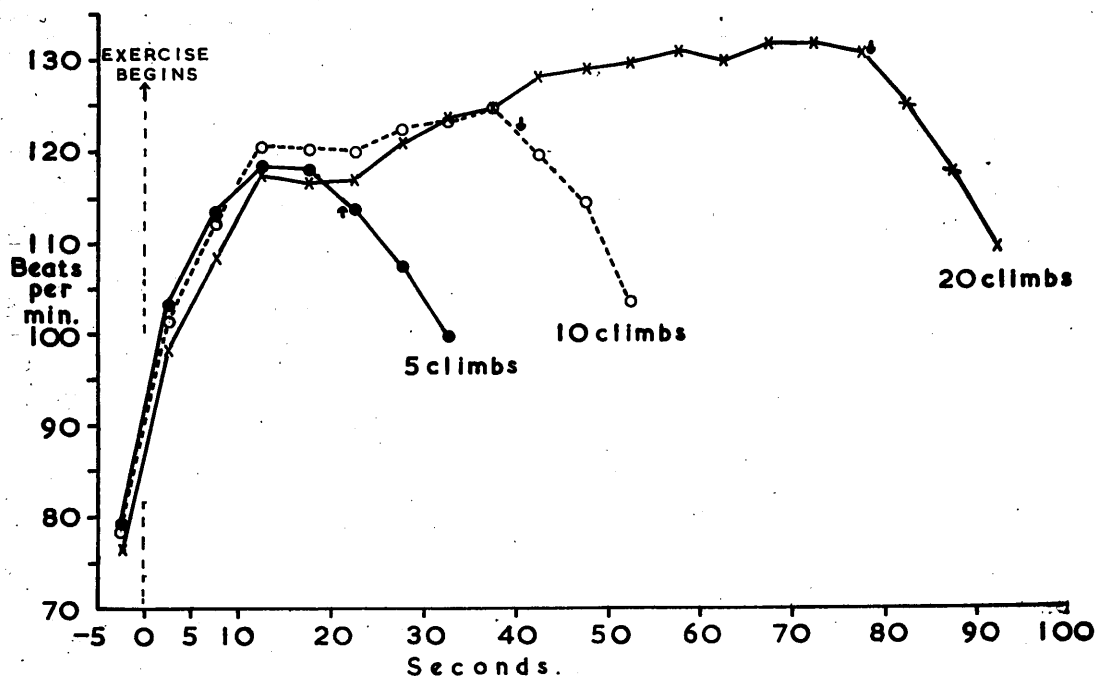


Fig. 22. Series D. Graphs of mean heart rates in 5 second intervals showing the effect of varying the duration of the exercise. The exercise consisted of 5, 10 and 20 ascents of the two steps at the standard rate of 96 steps per minute. The small arrows indicate the end of exercise for each curve.

EFFECT OF SPEED AND DURATION OF EXERCISE

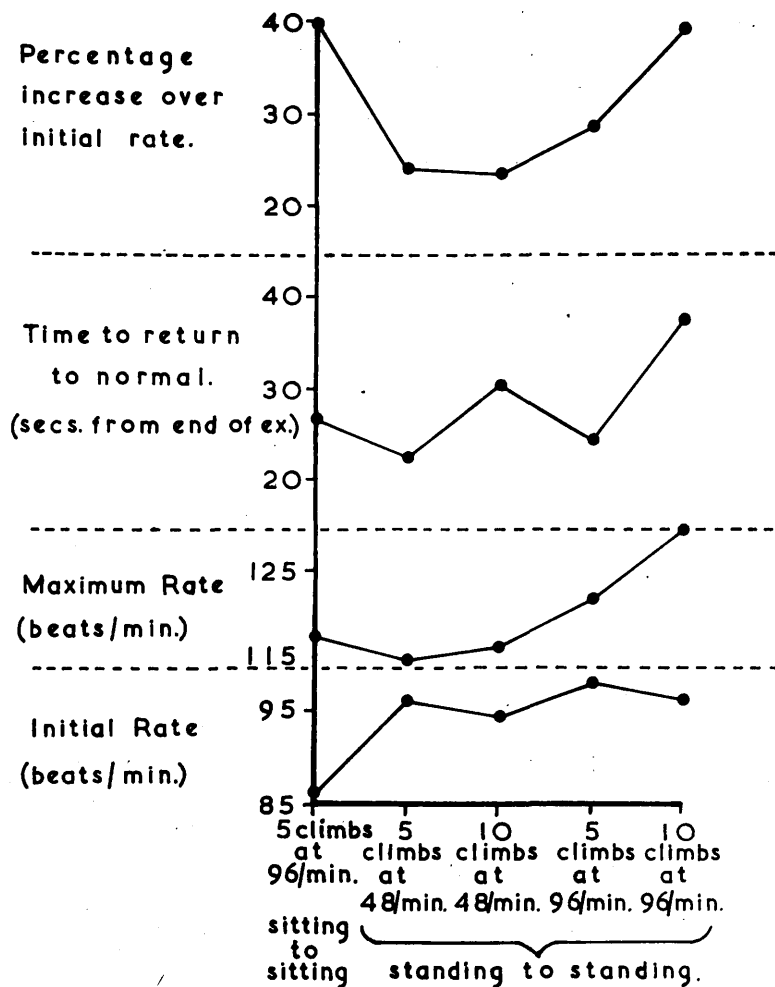


Fig. 23. Series S. The effect of varying the speed and duration of exercise on some heart rate indices.

The various rates of stepping and durations of exercise are indicated on the abscissa.

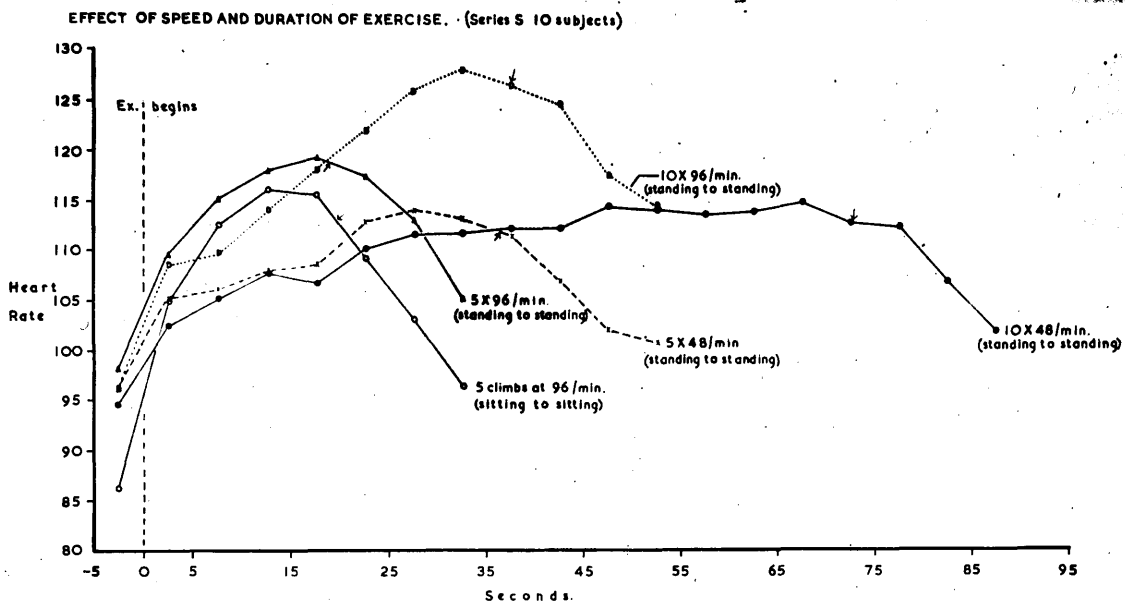


Fig. 24. Series S. Graphs of mean heart rates in 5 second intervals showing the effect of varying the speed and duration of exercise. The small arrows indicate the end of exercise for each curve.

Series B.N. 22 Subjects.

Heart Rate during Exercise in Normal Subjects.

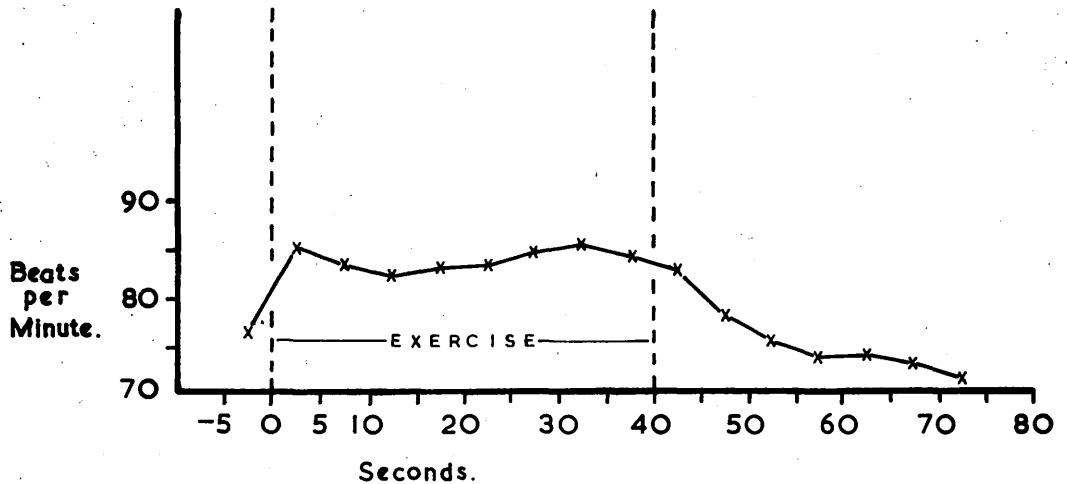


Fig. 25. Series B.N. (22 subjects). Graph of mean heart rates in successive 5 second intervals before, during and after the standard arm-stretching exercise performed in the recumbent posture.

Series C. V. D. H. Cases.

Heart Rate during Exercise in Tolerance Groups.

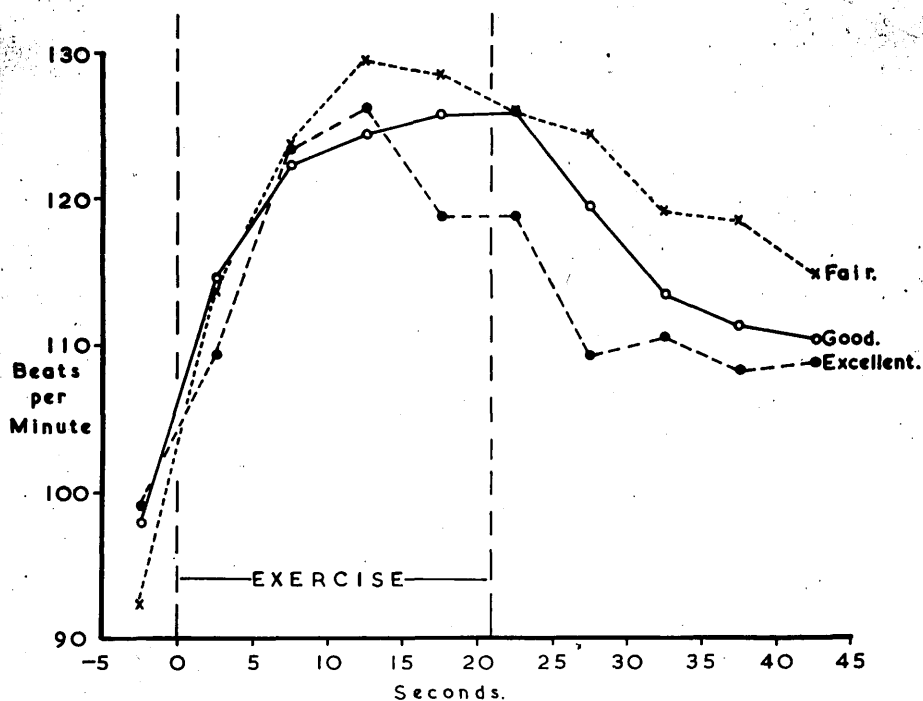


Fig. 26. Series C. V.D.H. Cases.

Graphs of the mean heart rates before, during and after the standard step test in patients classified into their different 'tolerance groups'.

Heart Rate during exercise in Normal Subjects.

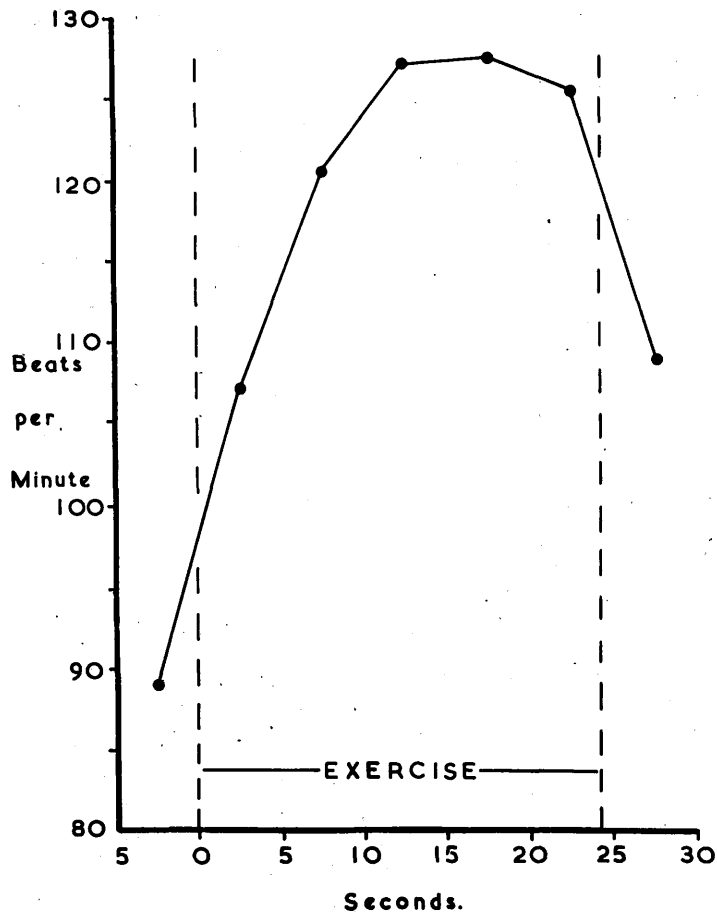


Fig. 27. Series A + Series B.

Graph of mean heart rate before, during and after the standard step test in 100 normal subjects.

This figure is graphed to the same scale as Fig. 26 and is for comparison with that figure.

Series C. Effect of Posture on Heart Rate in Normals and V D H Cases.

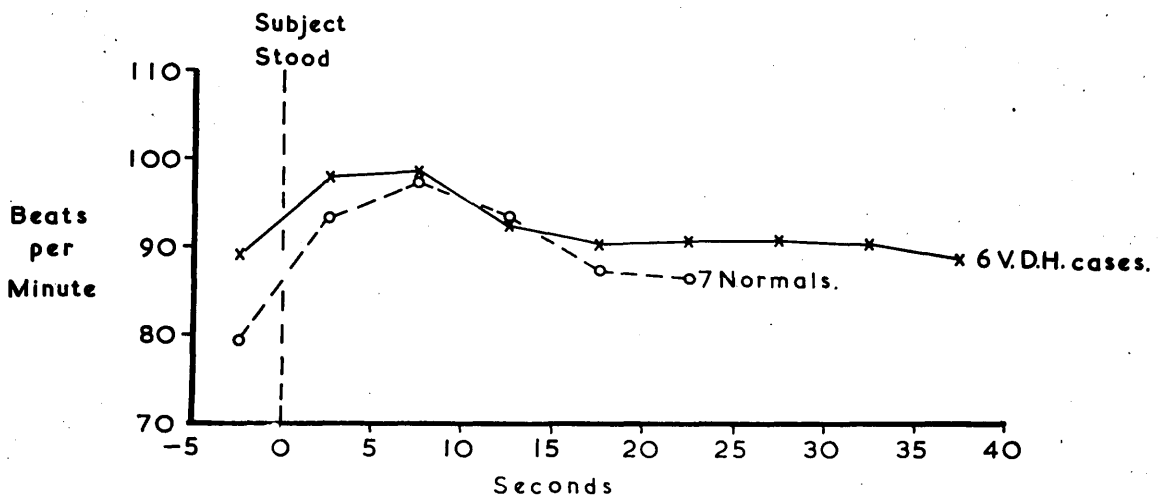


Fig. 28. Graphs of mean heart rates during change of posture from sitting to standing.

Upper graph (full line) - results in 6 cases of valvular disease.

Lower graph (broken line) - results in 7 normal subjects.

Series C Exercise Component only.

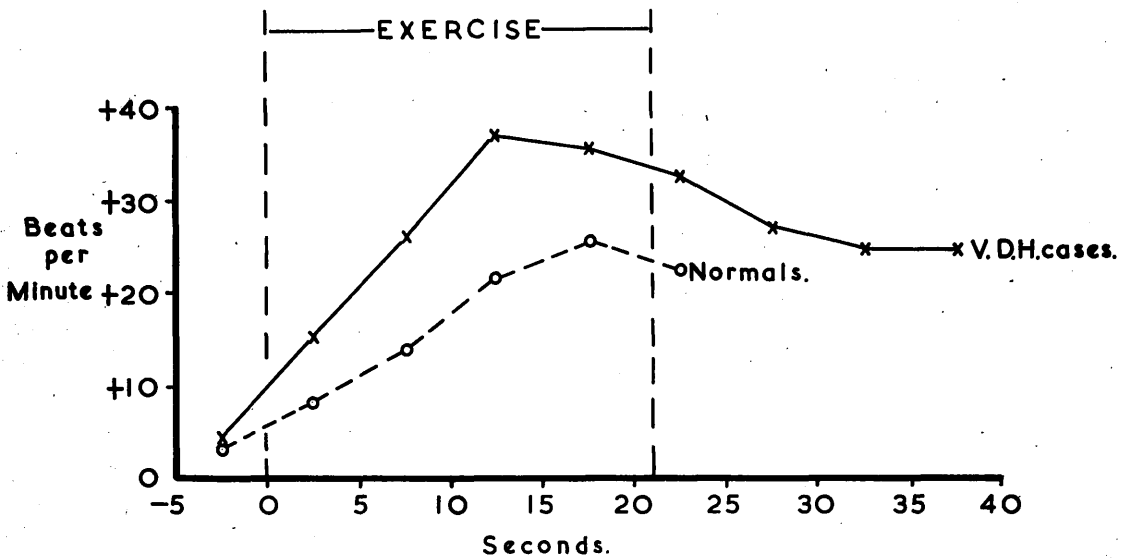


Fig. 29. Graphs of mean heart rates during the standard step test showing the effect of the exercise component only.

Upper graph (full line) - results in 6 cases of valvular disease.

Lower graph (broken line) - results in 7 normal subjects.

Series C. Exercise Component only. V.D.H. cases.

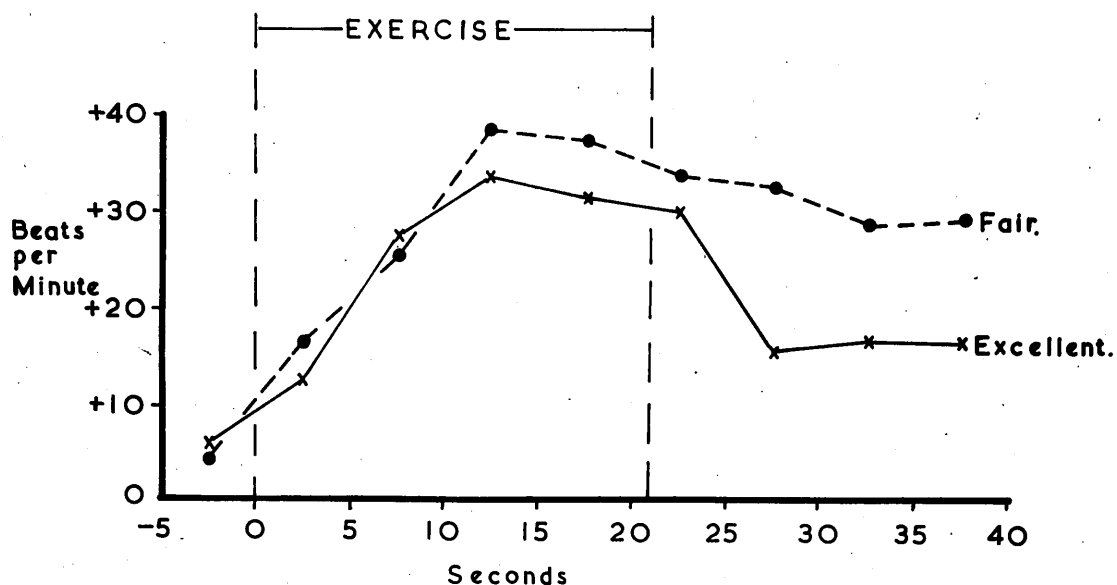


Fig. 30. Graphs of the effect of the exercise component of the step test on the heart rates of patients with valvular disease belonging to two different 'tolerance groups'.

Full line - mean heart rates in 2 patients with excellent tolerance.

Broken line - mean heart rates in 5 patients with fair tolerance.

Series B.N.

Bed exercise on heart rates of normals and patients.

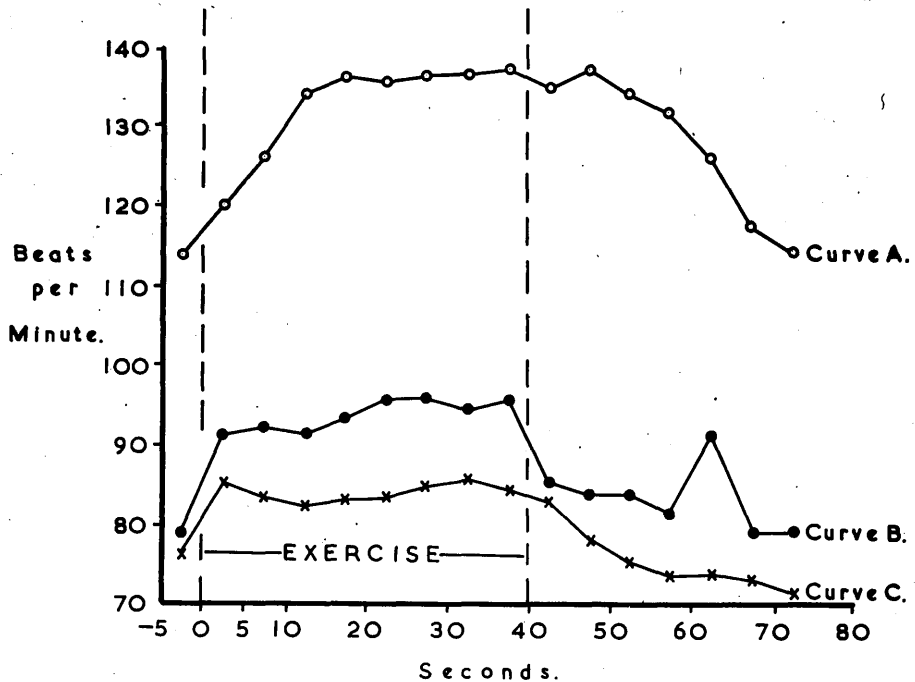


Fig. 31. Graphs of the effect of the arm-stretching exercise on the heart rates of normals and cardiac patients.

Curve A - mean curve of heart rate in 4 cases of valvular disease.

Curve B - curve of heart rate in convalescent V.D.H. patient.

Curve C - mean curve of heart rate in 22 normal subjects.

Series B.C. Effect of treatment on response to bed exercise.

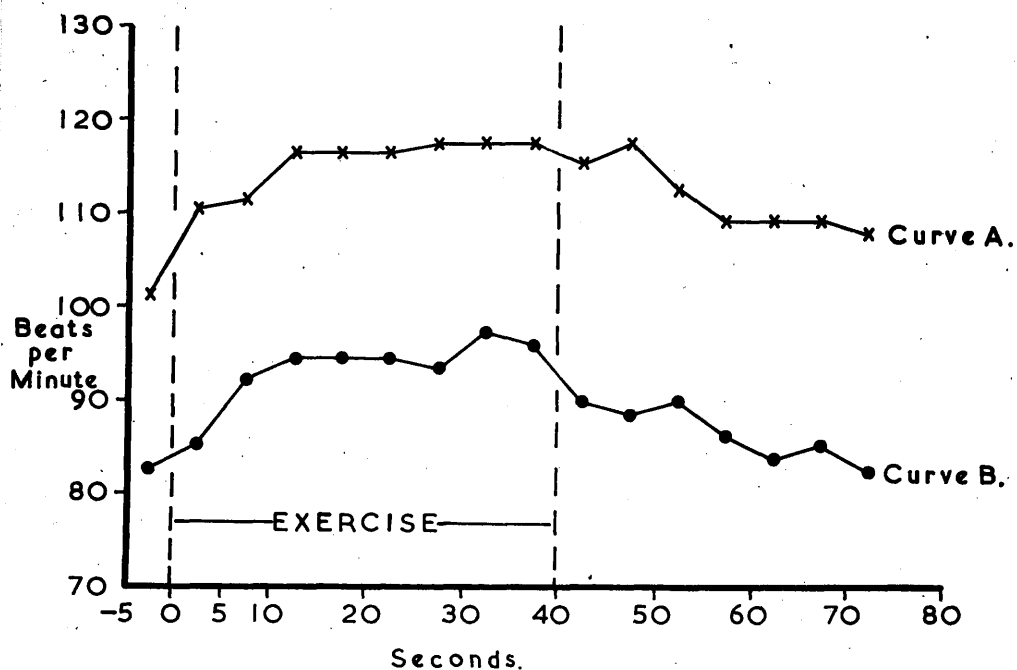


Fig. 32. Graphs of the effect of treatment on the response of the heart rate to the arm-stretching exercise.

Curve A - Exercise test performed shortly after admission to hospital.

Curve B - Exercise test performed by same patient 6 weeks later.

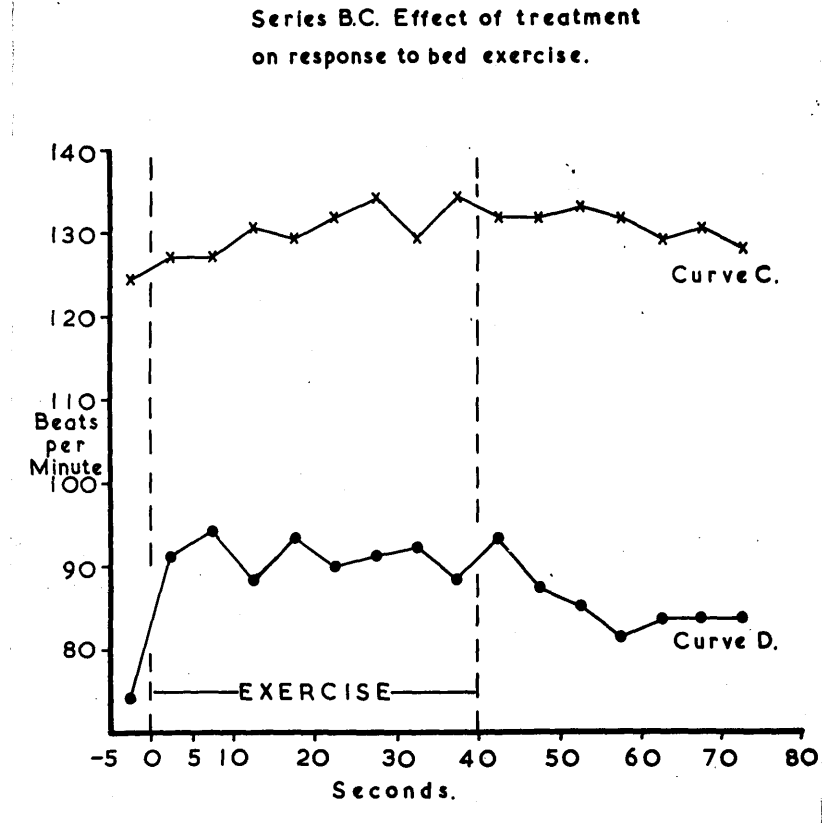


Fig. 33. Graphs of the effect of treatment on the response of the heart rate to the arm-stretching exercise.

Curve C - Exercise test performed 6 days after admission to hospital.

Curve D - Exercise test performed by the same patient two weeks later.

Series C

Heart Rates during standard exercise
in Angina Pectoris Cases.

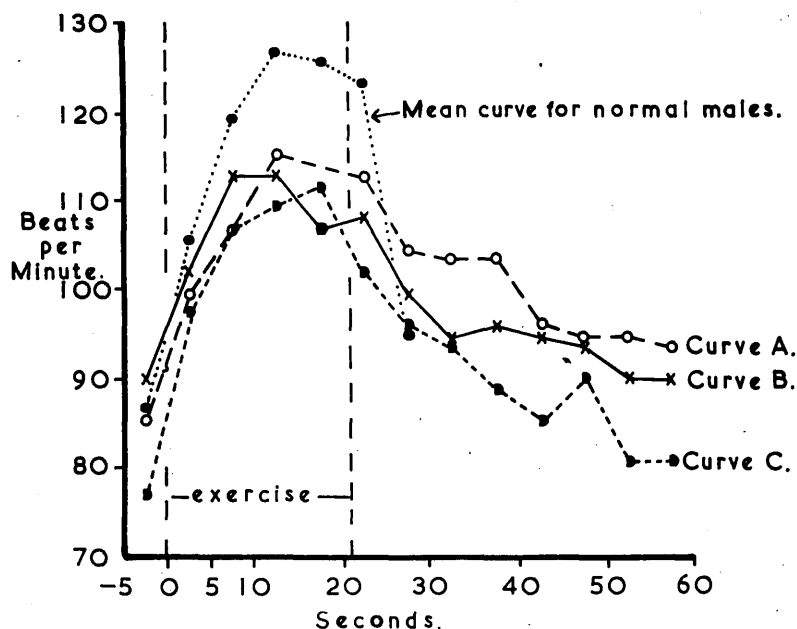


Fig. 34. Graphs of heart rate during the standard two-step exercise in normal males and in three patients with angina pectoris.

Upper Curve (dotted line) - mean result for normal males (Series A)

Curve A - heart rate in anginal patient with fair tolerance.

Curve B - heart rate in anginal patient with poor tolerance.

Curve C - heart rate in anginal patient with fairly good tolerance.

Fig. 35. Upper tracing - Kymograph record showing burst of ventricular extrasystoles in a patient with angina pectoris. These occurred when the subject was suddenly spoken to.

Lower tracing - Electrocardiogram (lead IVF) from this subject, showing that the extrasystoles arose from both ventricles.

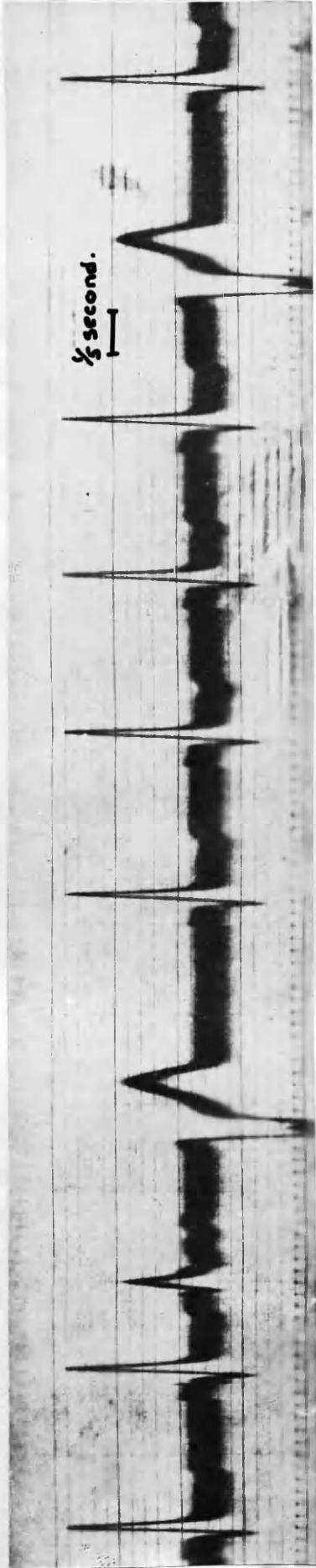
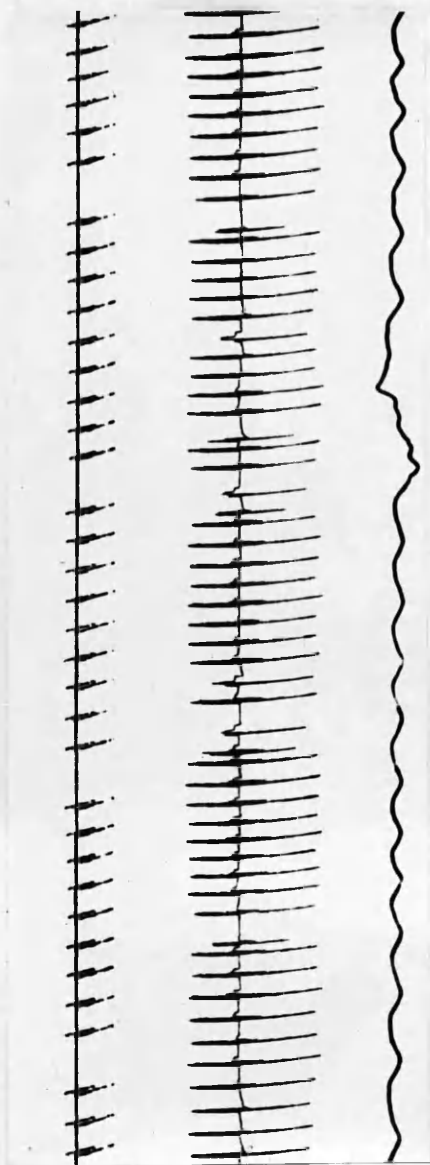


Fig. 35.

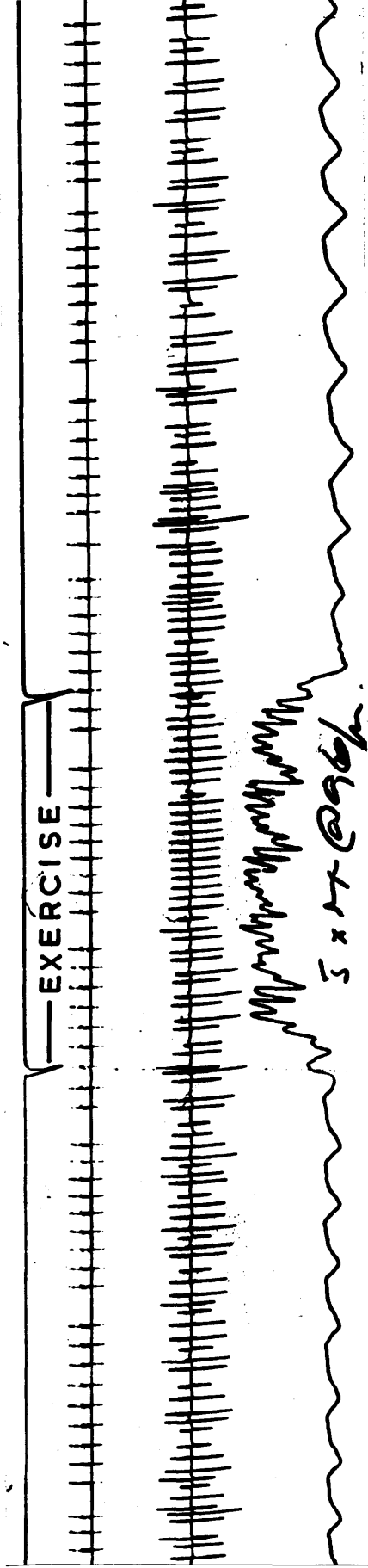


Fig. 36. Kymograph tracing showing heart beats before, during and after exercise in a case of coronary thrombosis.

The standard two-step test was performed between the dips in the signal line.

Multiple ventricular extrasystoles are seen before and after the exercise, but disappear for a short time towards the end of the step test. There is a marked tendency to coupling of beats, especially after exercise.

Series C. Heart Rate during exercise in cases of
Effort Syndrome and Coronary Thrombosis.

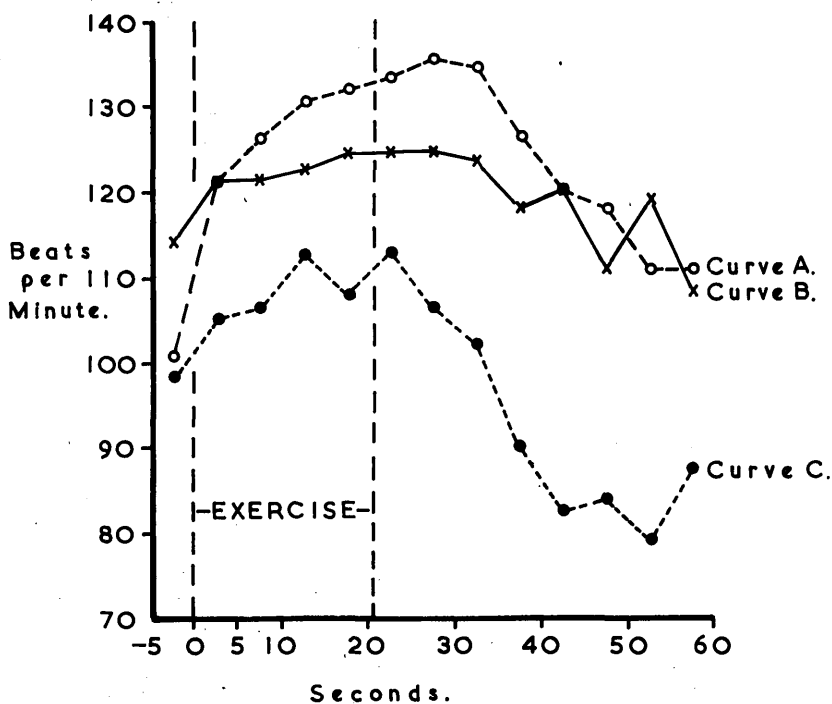


Fig. 37. Graphs of heart rate during the standard two-step exercise in cases of effort syndrome and coronary thrombosis.

Curve A - heart rate in effort syndrome case with good tolerance.

Curve B - heart rate in effort syndrome case with fairly good tolerance.

Curve C - heart rate in a case of coronary thrombosis.

Series C. and B.C. Heart Block Cases.

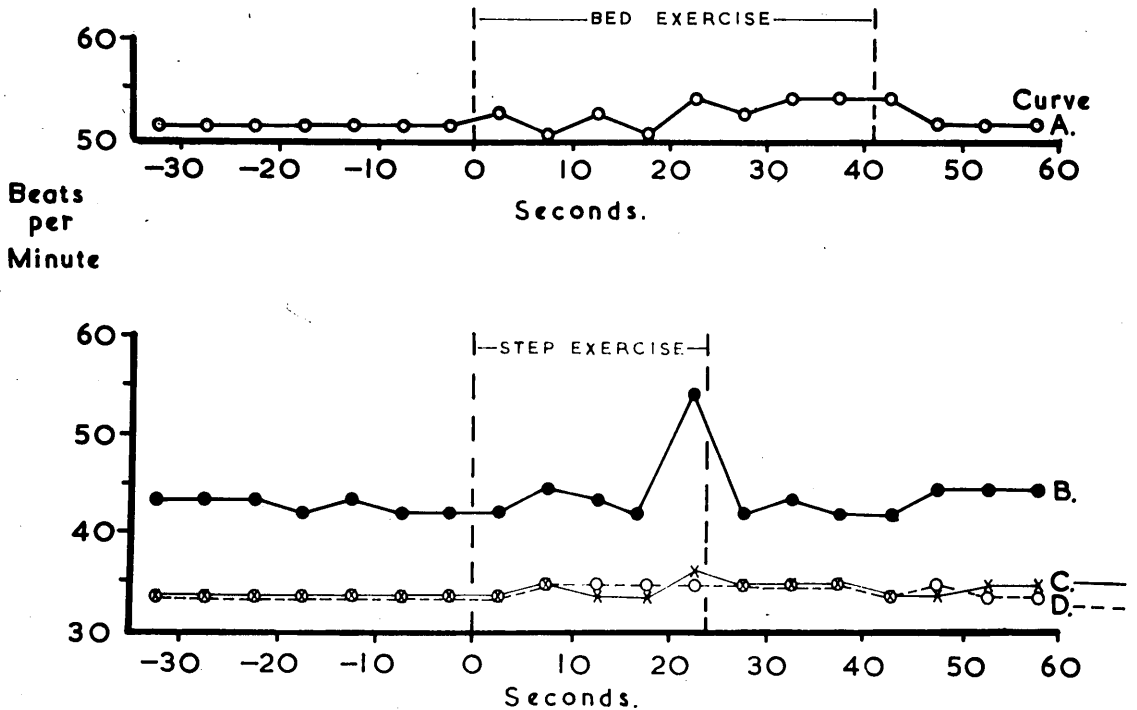


Fig. 38. Graphs of heart rate before, during and after exercise in cases of heart block.

Curve A - Effect of arm-stretching exercise on the heart rate in a case of partial heart block.

Curve B - Effect of standard step test on the heart rate in a case of partial heart block.

Curve C - Effect of standard step test on the heart rate of a patient with complete heart block. (Same patient as in Curve D, but taken one year later).

Curve D - Effect of standard step test on the heart rate of a patient with complete heart block.

Case C35. Heart Block.

Heart Rate during standard and prolonged exercise.

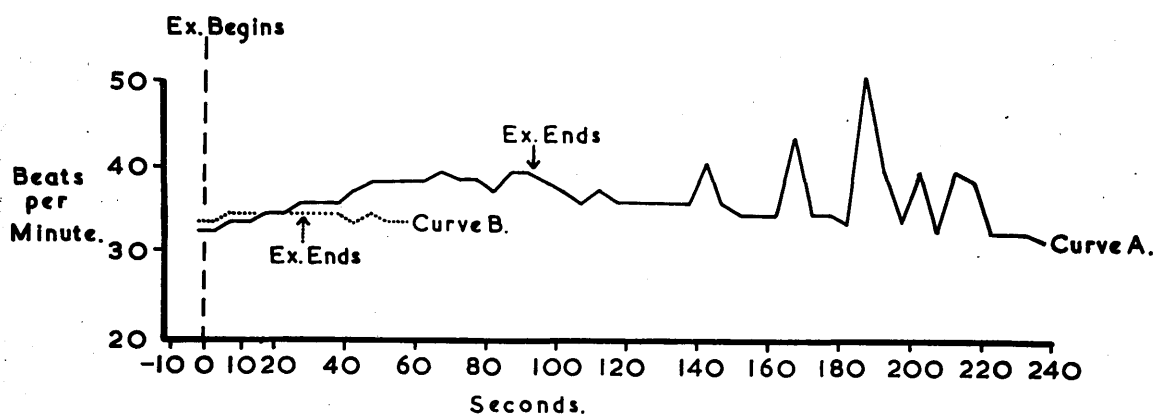


Fig. 39. Graphs of heart rate during and after step tests in a case of complete heart block.

Curve A - heart rate during and after a prolonged step test (20 ascents).

Curve B - heart rate during and after the standard step test (5 ascents).

In each case the end of exercise is indicated by the arrow.

Fig. 40. Kymograph tracing of heart beats of subject with complete heart block (Case C 35). This tracing was taken some time after prolonged exercise (20 ascents of the two steps) and shows recurrent ventricular extrasystoles.

Upper line -- time in seconds.
Middle line - heart beats.
Lower line -- respiration.

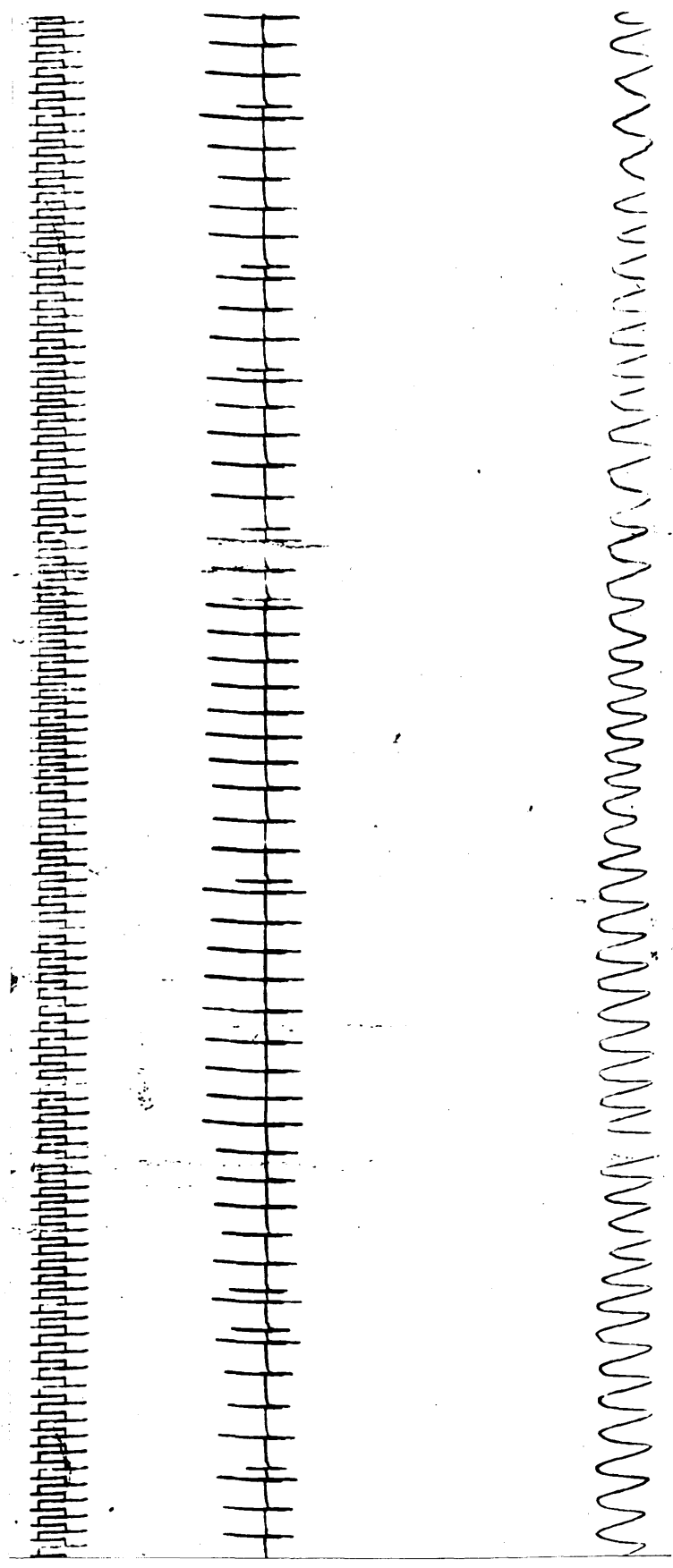


Fig. 40.

Series B.N.

Heart Rate during Exercise in Case of Hyperthyroidism.
Effect of Treatment.

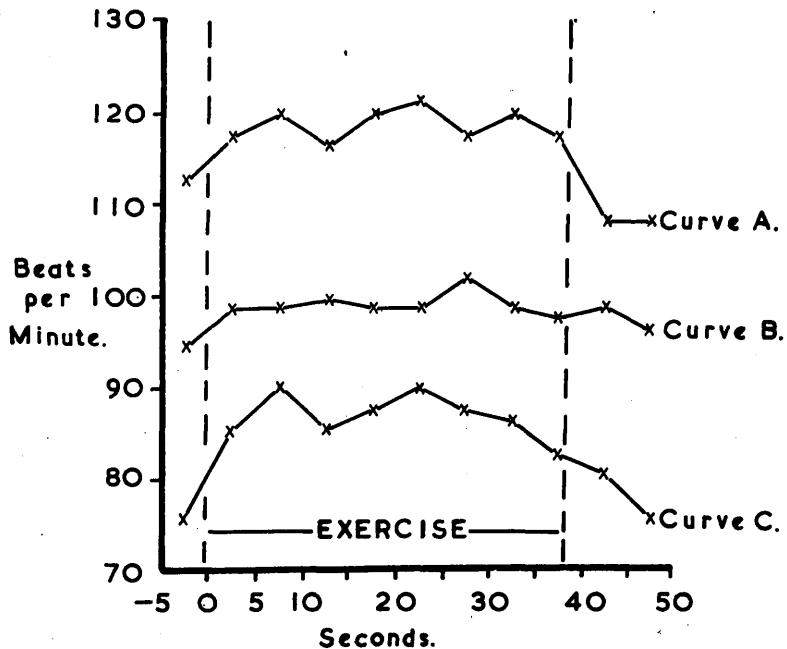


Fig. 41. Graphs showing the effect of treatment on the response of the heart rate to arm-stretching exercise in a case of hyperthyroidism.

Curve A - response of untreated patient.

Curve B - response after treatment with iodine.

Curve C - response after partial thyroidectomy.

Series B.C. Heart Rate during exercise in a case of Hyperthyroidism before and after operation.

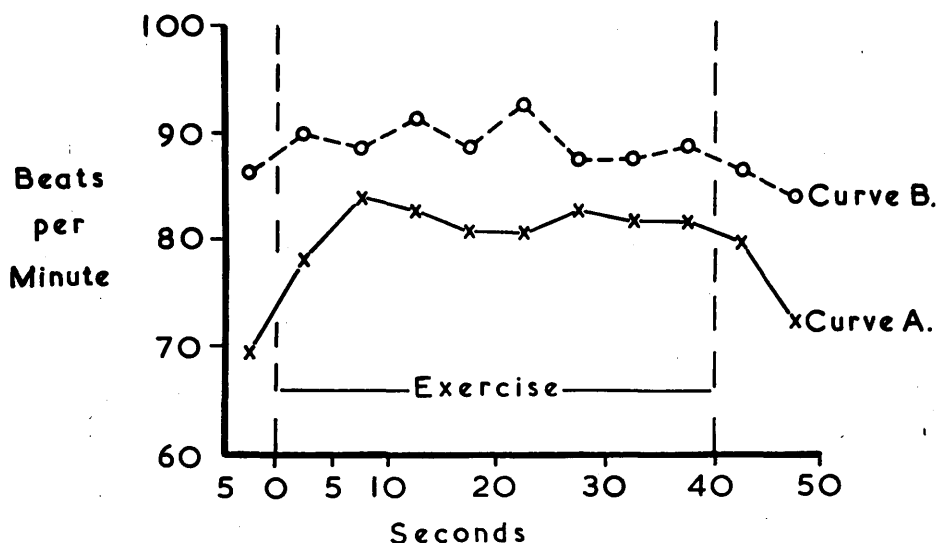


Fig. 42. Graphs showing the effect of treatment on the response of the heart rate to arm-stretching exercise in a case of hyperthyroidism.

Curve A - response of untreated patient.

Curve B - response after partial thyroidectomy.

Series C. Auricular Fibrillation Cases on Digitalis.

Heart Rate during Exercise by Tolerance Groups.

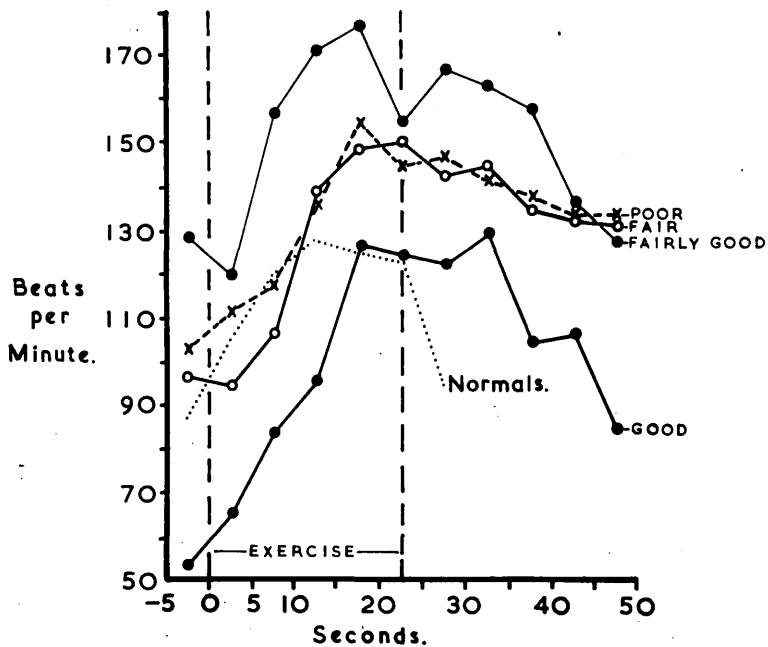


Fig. 43. Series C. Auricular fibrillation patients on digitalis.

Graphs of mean heart rates before, during and after the standard step test in patients classified into their different 'tolerance groups'.

The dotted line shows the mean curve for normal subjects.

Series C. Heart Rate during exercise in cases of
Auricular Fibrillation not on Digitalis.

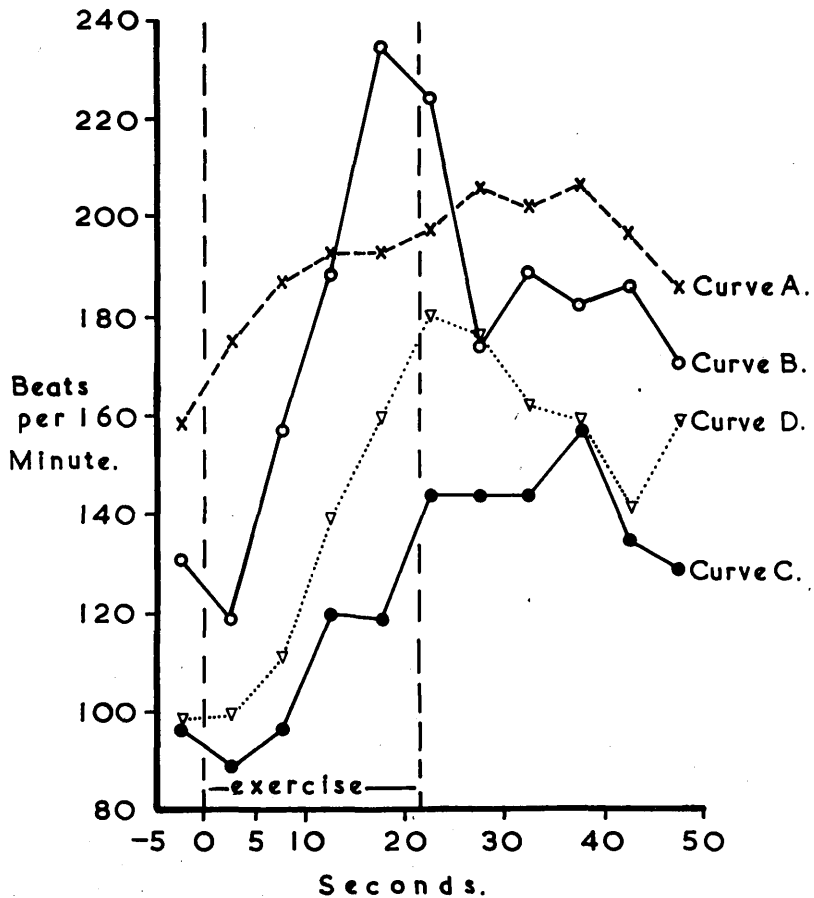


Fig. 44. Series C. Auricular fibrillation patients not on digitalis.

Graphs of heart rate before, during and after the standard step test in four patients.

Curves A, B, C and D are each from a separate patient.

EXERCISE

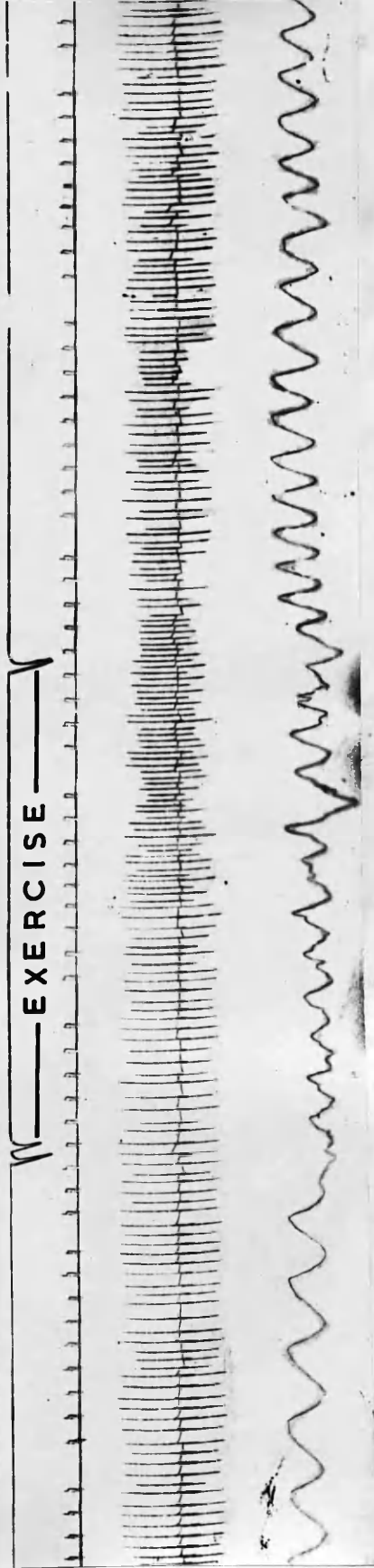


Fig. 45. Kymograph tracing of heart beats from a patient with auricular fibrillation. Case C 2. Between the dips in the signal line the patient performed the standard two-step test. During the last half of exercise the extremely high heart rate of 235 beats per minute was attained.

Upper line - signal
Second line - time in seconds
Third line - heart beats
Fourth line - respiration

Series B.C.

Heart Rate during Exercise in
Two Cases of Auricular Fibrillation.

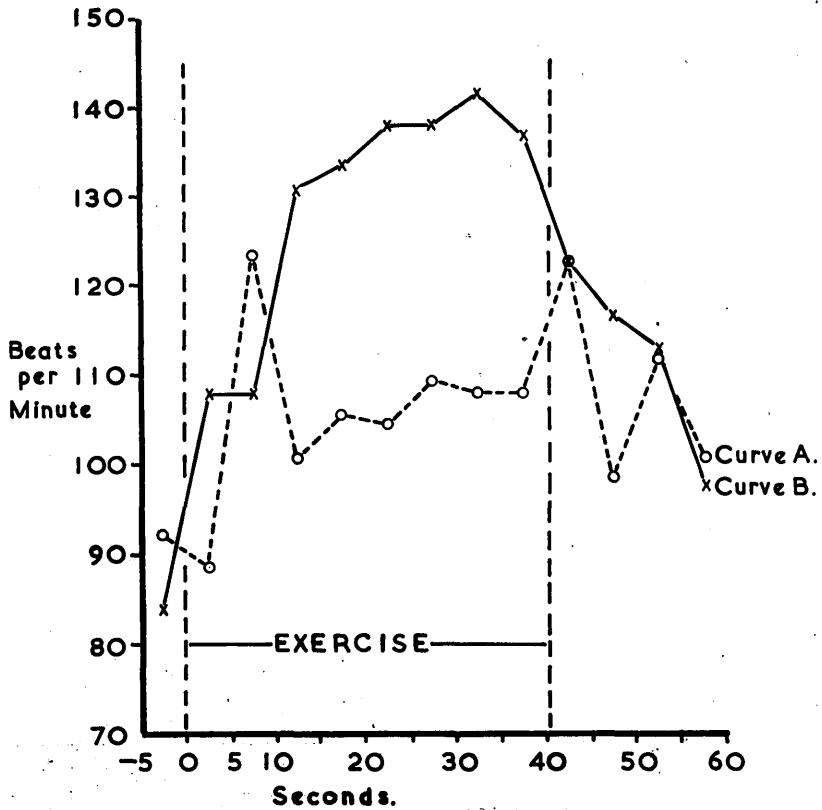


Fig. 46. Heart rate during arm-stretching exercise in two cases of auricular fibrillation.

Curve A - Case B.C. 27, not on digitalis.

Curve B - Case B.C. 26, on digitalis.

Series C and B.C. Step and Bed exercise in the same
fibrillation patient compared with normals.

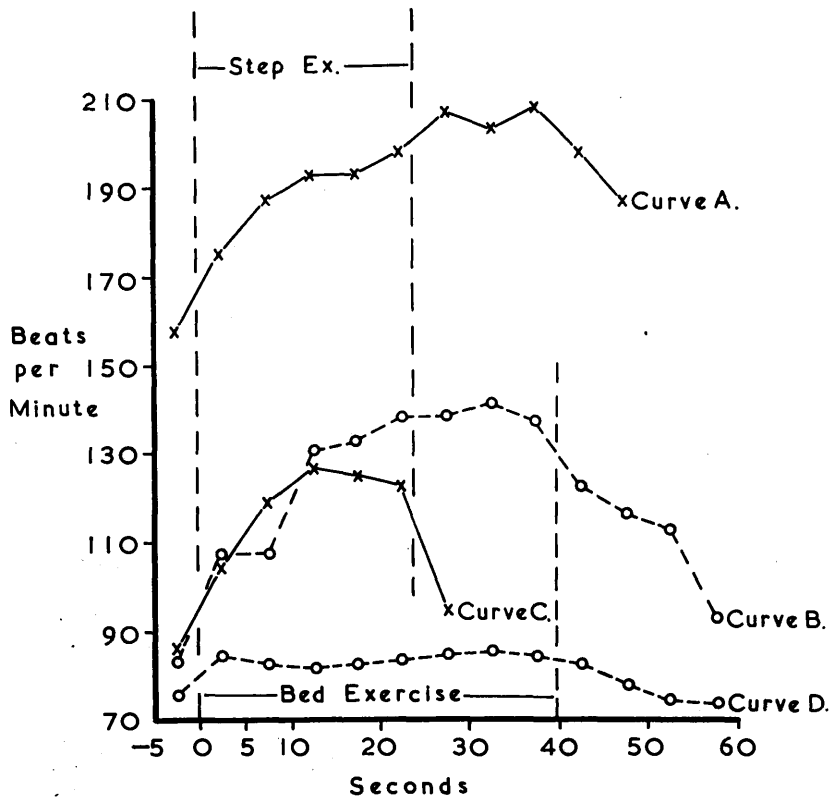


Fig. 47. Effects of the standard step and arm-stretching exercises on the heart rate of the same auricular fibrillation patient, with normal curves for comparison.

Curve A - effect of step test on patient's heart rate

Curve B - effect of arm-stretching on patient's heart rate.

Curve C - effect of step test on heart rate of normal subjects.

Curve D - effect of arm-stretching on heart rate of normal subjects.

Series C. Case C49.

Auricular Fibrillation on Digitalis.

Standard Exercise 3 times at 15 minute intervals.

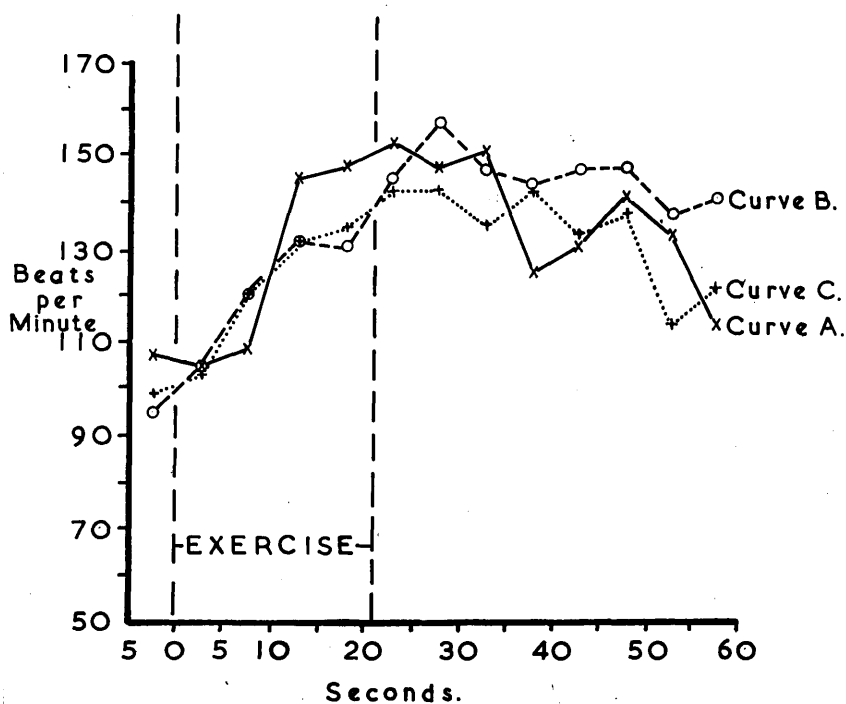


Fig. 48. Effect of repetition of the standard step test at 15 minute intervals on the heart rate of a patient with auricular fibrillation.

Curve A - Heart rate before, during and after the standard step test.

Curve B - the same, 15 minutes later.

Curve C - the same, 15 minutes after Curve B.

Series C. Effect of clinical condition on Heart Rate during exercise in two cases of Auricular Fibrillation.

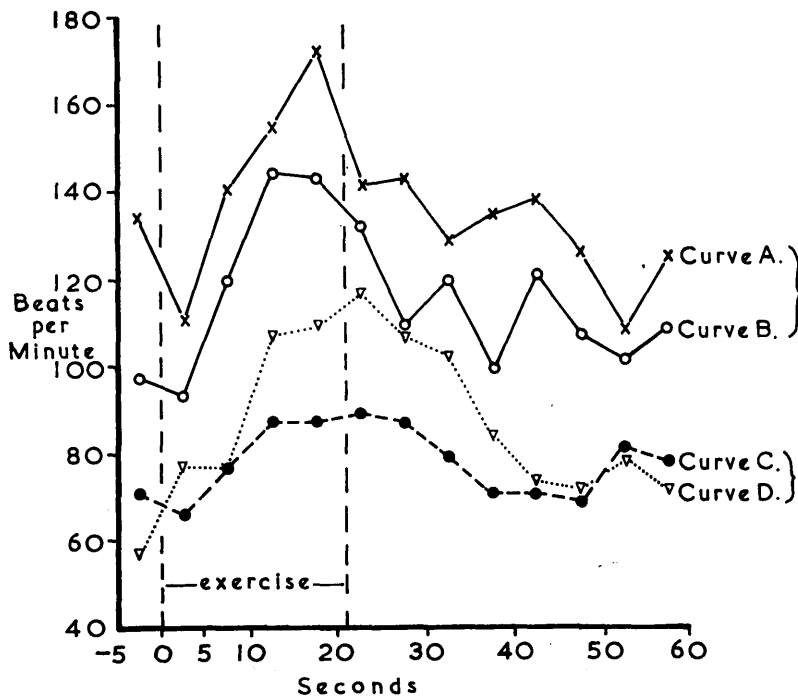


Fig. 49. The effect of clinical condition on the heart rate during the standard step test in two cases of auricular fibrillation.

Curve A - heart rate before, during and after the step test.

Curve B - the heart rate in the same patient one year later, when the clinical condition had improved.

Curve C - heart rate before, during and after the step test in another fibrillation patient.

Curve D - the heart rate six months later in the same patient as curve C, when the clinical condition had deteriorated.

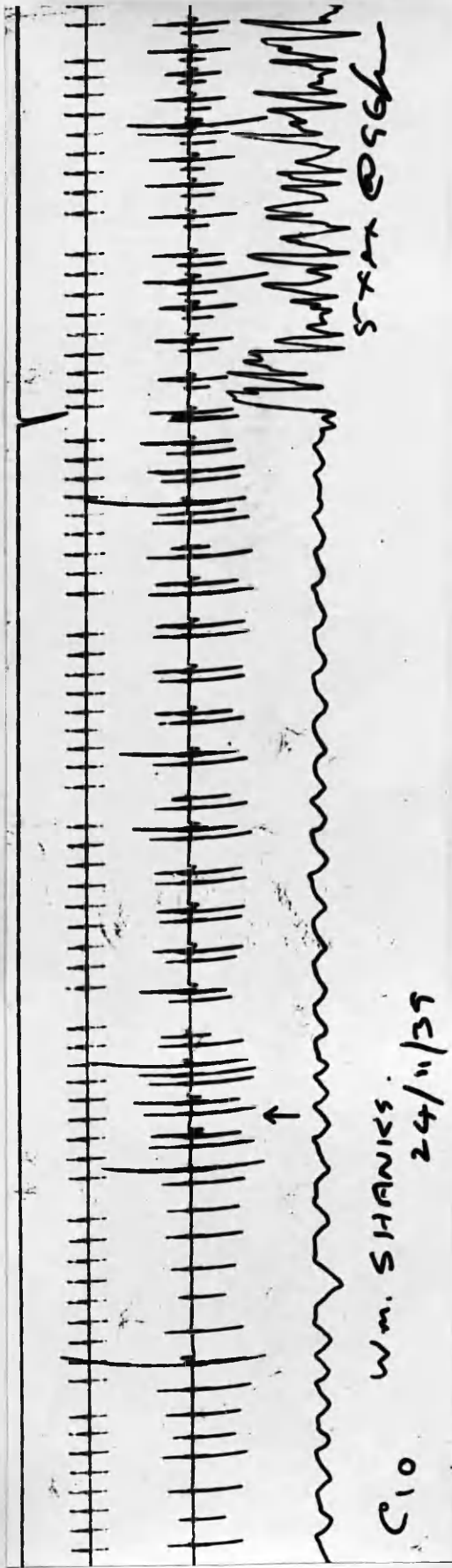


Fig. 50. Kymograph tracing showing onset of a period of coupling in a patient with auricular fibrillation. (Case C 10).

The coupled beats begin after a burst of extrasystoles indicated by the arrow.

The dip in the signal line indicates the beginning of the standard step test.

Series B.C. Effect of Treatment on Fibrillation Case.

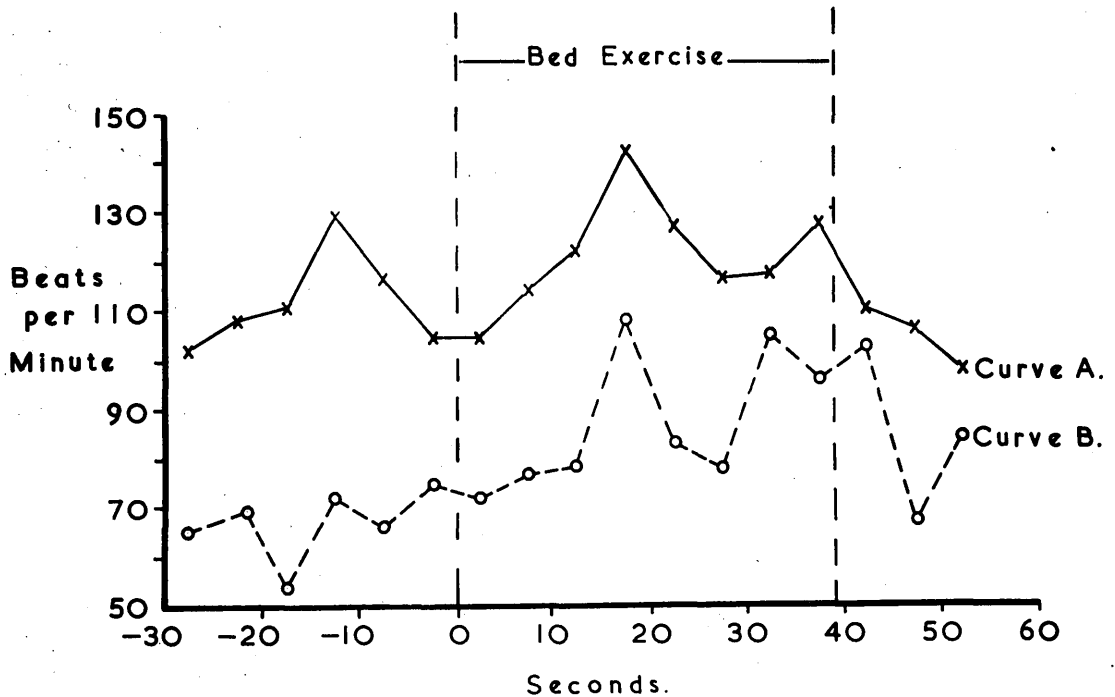


Fig. 51. Case B.C. 21. Effect of treatment on the heart rate during the arm-stretching exercise in a patient with fibrillation.

Curve A - curve taken three weeks after admission.
On digitalis.

Curve B - taken two weeks after curve A.

For details of treatment see script.

Series B.C. Effect of Treatment on Fibrillation Case. (continued).

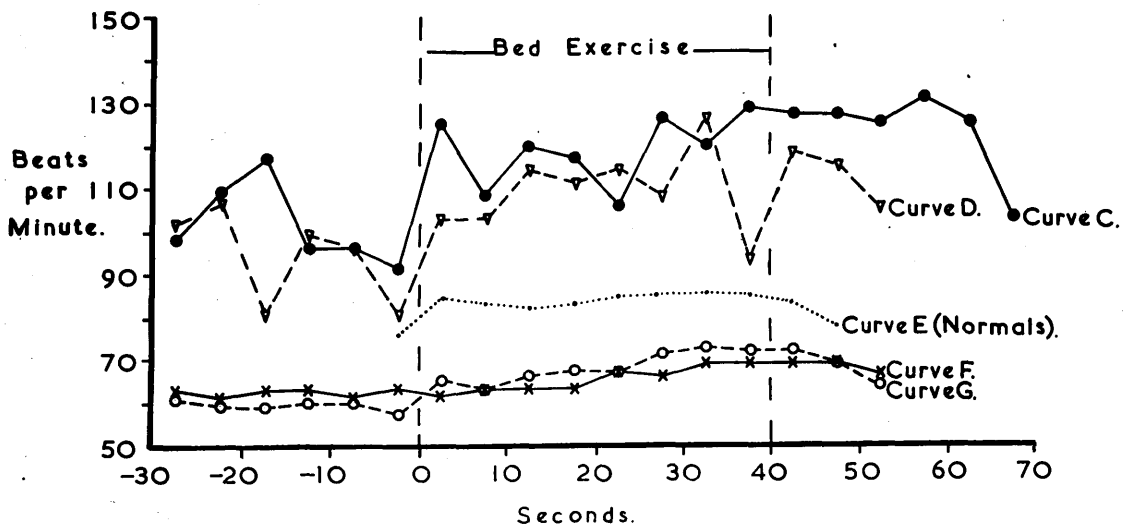


Fig. 52. Case B.C. 21. (continued from Fig. 51).

Curve C - taken one week after curve B and 12 hours after quinidine.

Curve D - taken 3 days after curve C and immediately after quinidine.

Curve E - mean curve of normal subjects for comparison.

Curve F - taken 5 days after curve D and one hour after return to normal rhythm following quinidine.

Curve G - taken 3 days after curve F. No further medication.

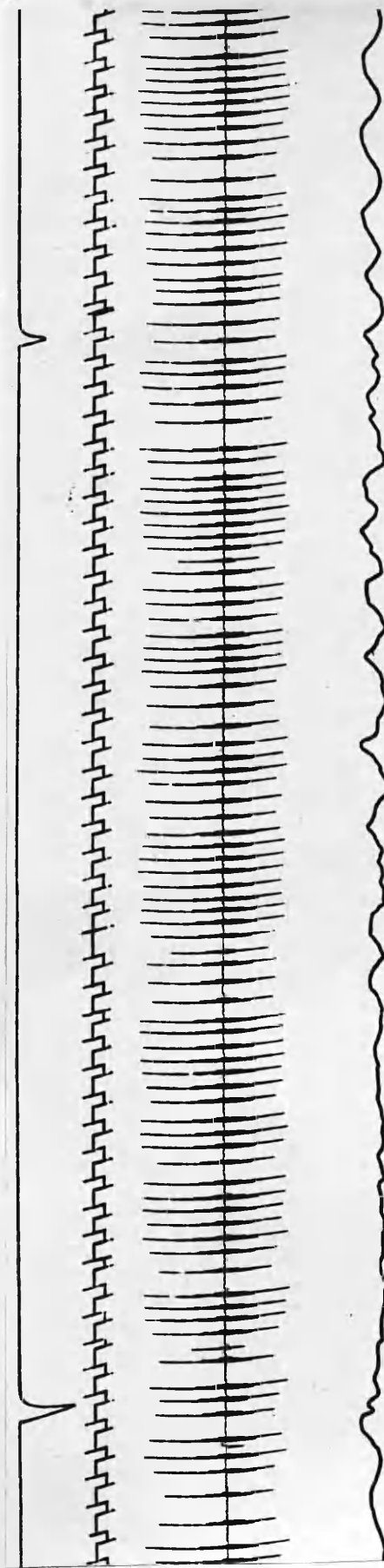
For details of treatment see script.

Fig. 53. Kymograph tracings of the heart rate throughout arm-stretching exercise in a case of auricular fibrillation before and after successful treatment with quinidine.

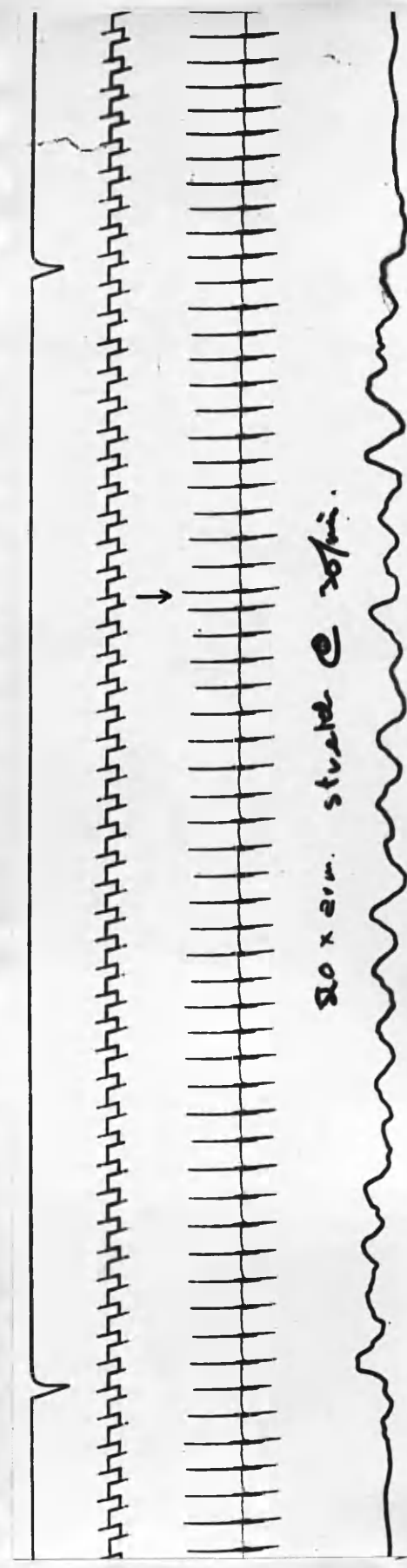
Upper tracing - before restoration of normal rhythm.

Lower tracing - after return to normal rhythm. An auricular extrasystole occurs at the arrow.

In both tracings the patient performed the standard arm-stretching exercise between the dips in the signal line.



20x20m stretch @ 20fps.



20x20m stretch @ 20fps.

Fig. 53.

Fig. 54. Kymograph tracings of heart beats during standard step-test in four patients with auricular fibrillation and in one normal subject.

Upper four tracings - auricular fibrillation patients.

Lowest tracing - normal subject.

In all four fibrillation patients there is a sudden delayed acceleration of the heart rate at the point indicated by the arrow in each case.

The normal subject shows no evidence of such a delayed acceleration.

In each case the standard step-test was performed between the dips on the signal line.

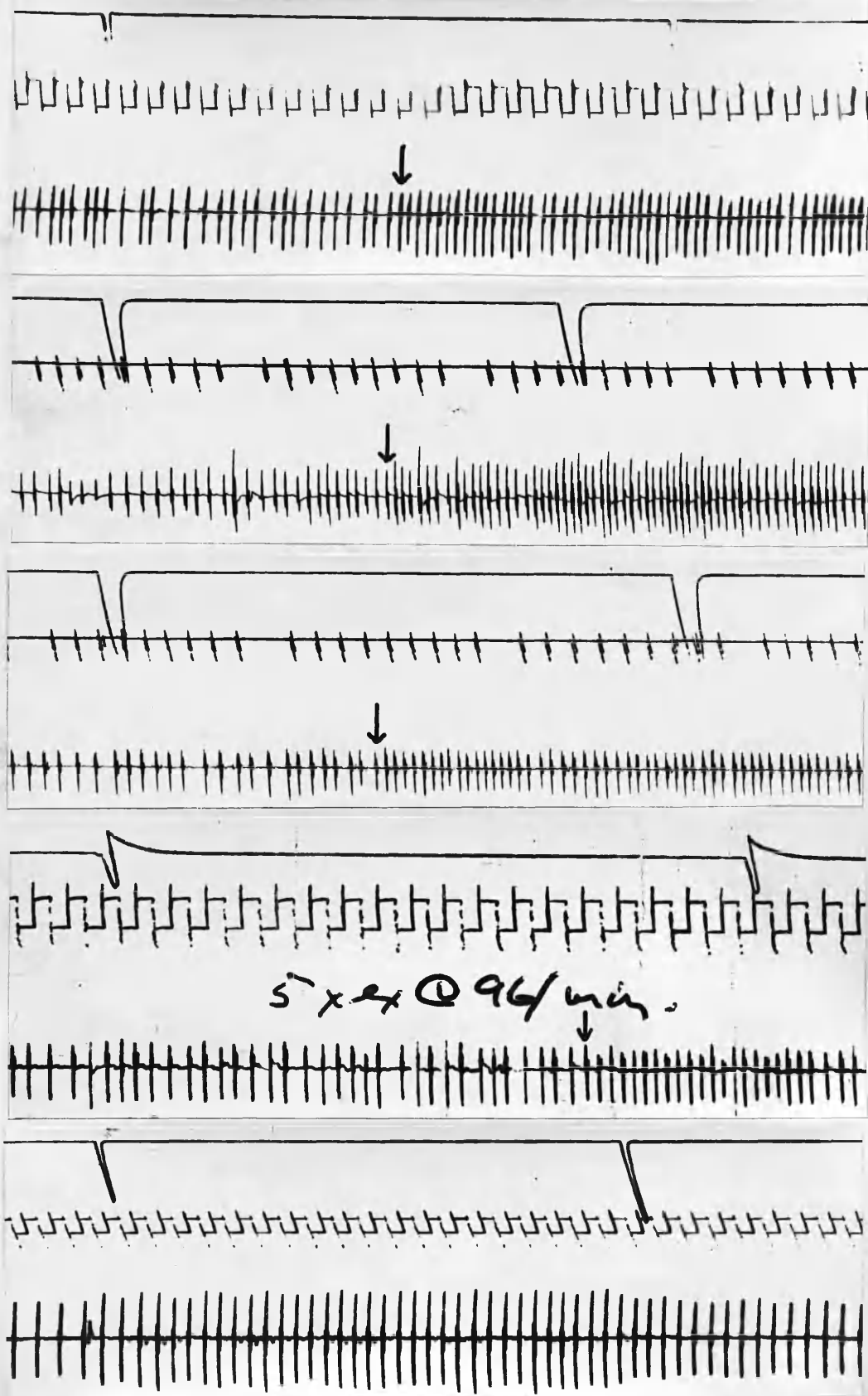


Fig. 54.

Series C. Intervals between beats during exercise in a case of auricular fibrillation.

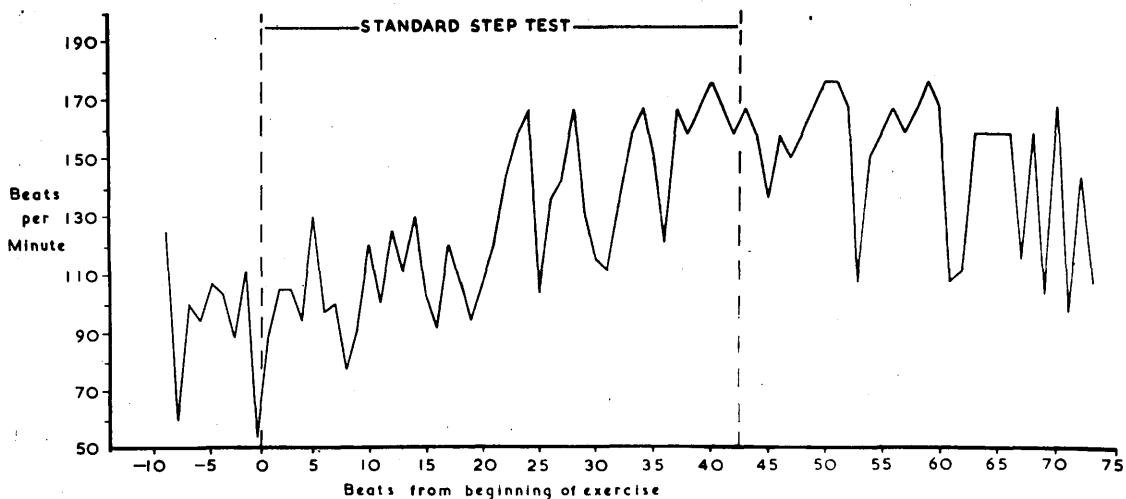


Fig. 55. Subject C 46, auricular fibrillation.

Graph showing intervals between successive heart beats before, during and after the standard two-step test.

Abscissa - individual beats from beginning of exercise.

Ordinate - interval between successive beats, expressed as beats per minute.

Fig. 56. Kymograph tracing of heart beats before and during exercise in a case of auricular fibrillation (Case C 46). This tracing was taken simultaneously with the electrocardiogram from which fig. 55 was calculated, and shows that during the resting period before exercise no sudden acceleration occurred comparable with that which took place during the exercise.

The commencement of exercise (standard step test) is indicated by the vertical ink line.

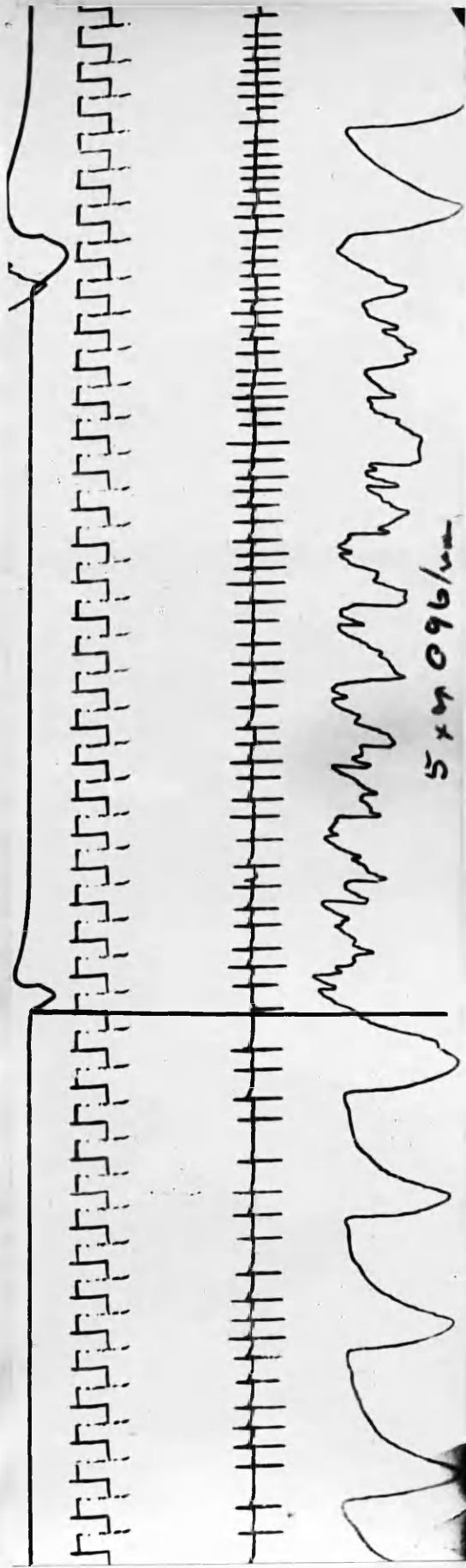


Fig. 56.

Fig. 58. Kymograph tracing of heart beats before and during exercise in a case of auricular fibrillation (Case C 40). This tracing was taken simultaneously with the electrocardiogram from which fig. 57 was calculated, and shows that bursts of rapid heart rhythm (up to 200 beats per minute) occurred during the resting period before exercise began.

The commencement of exercise (standard step test) is indicated by the vertical ink line.

Upper line - signal
Middle line - time in seconds
Lower line - heart beats.

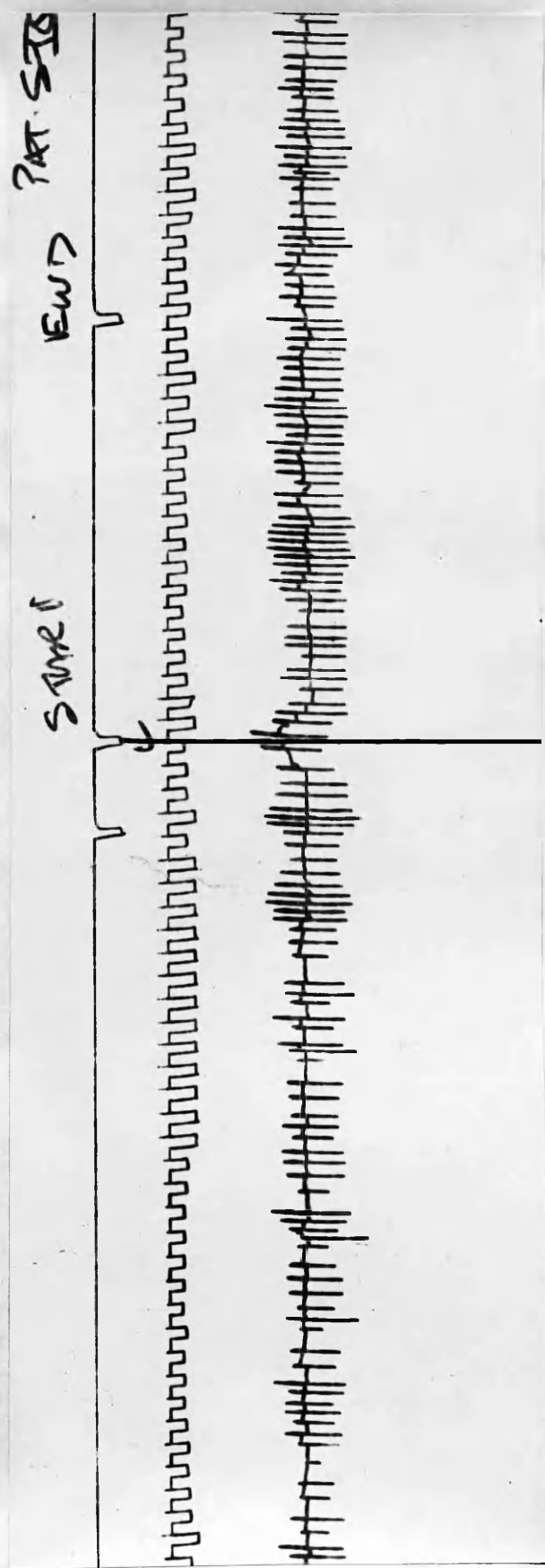


Fig. 58.

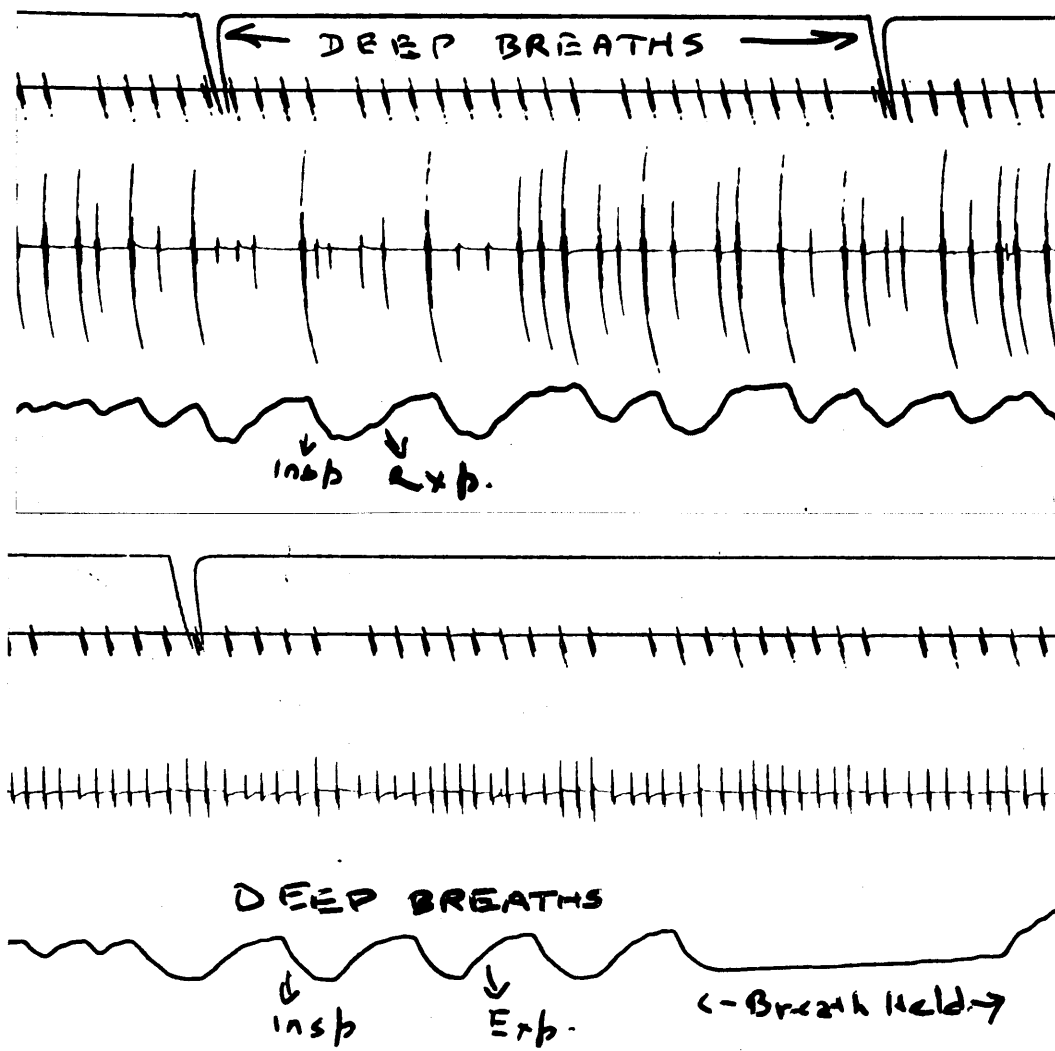


Fig. 59. Kymograph tracings showing the effect of deep breathing on the rhythm of the heart in two cases of auricular fibrillation. No consistent variation in rhythm can be seen.

Upper line - signal

Second line- time in seconds

Third line - heart beats

Fourth line- respiration (fall of pointer indicates inspiration)

Fig. 60. Electrocardiograms (string galvanometer) from a case of auricular fibrillation, (Case C 46).

- A - lead I, before exercise.
- B - lead II, before exercise.
- C - lead III, before exercise.
- D - lead I, immediately before standard step test.
- E - lead I, during last few seconds of standard step test showing onset of delayed acceleration at arrow.

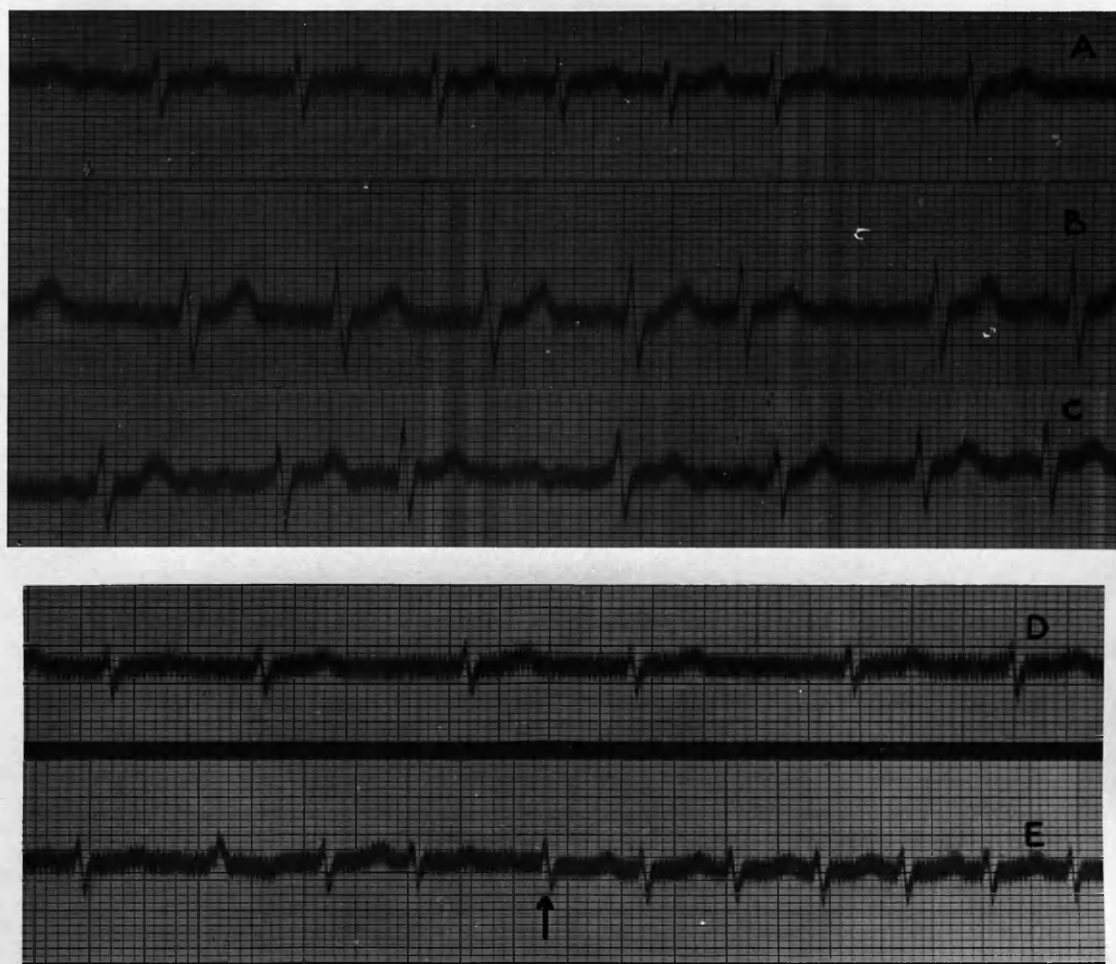


Fig. 60.

Fig. 61. Electrocardiograms (Matthews' oscillograph, chest leads) from a case of auricular fibrillation (Case C 40). Taken during exercise towards the end of the standard step test in each case.

Upper curve - small coupling condensers in amplifier.
Lower curve - large coupling condensers in amplifier.

The onset of delayed acceleration is indicated by an arrow in each case.

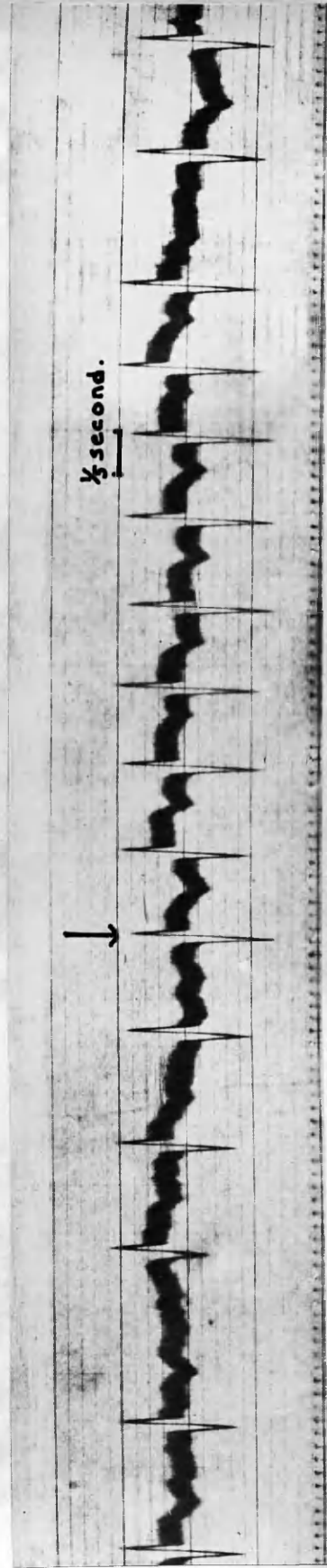


Fig. 61.

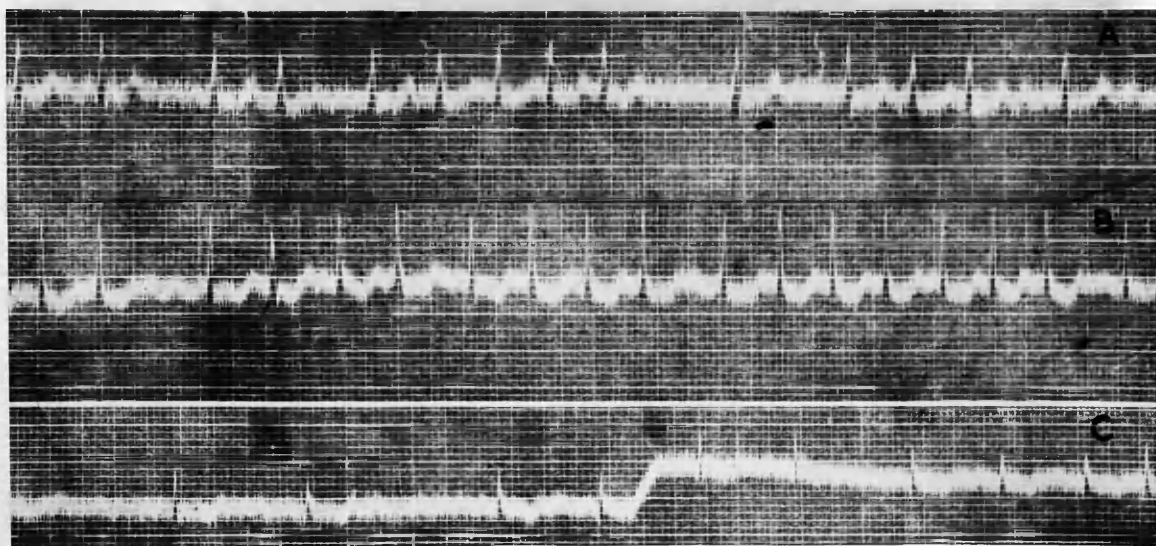


Fig. 62. Electrocardiograms (string galvanometer) from a case of auricular fibrillation. (Case C 40).

- A - lead I, before exercise.
- B - lead I, immediately after exercise.
- C - lead I, 5 minutes after exercise.

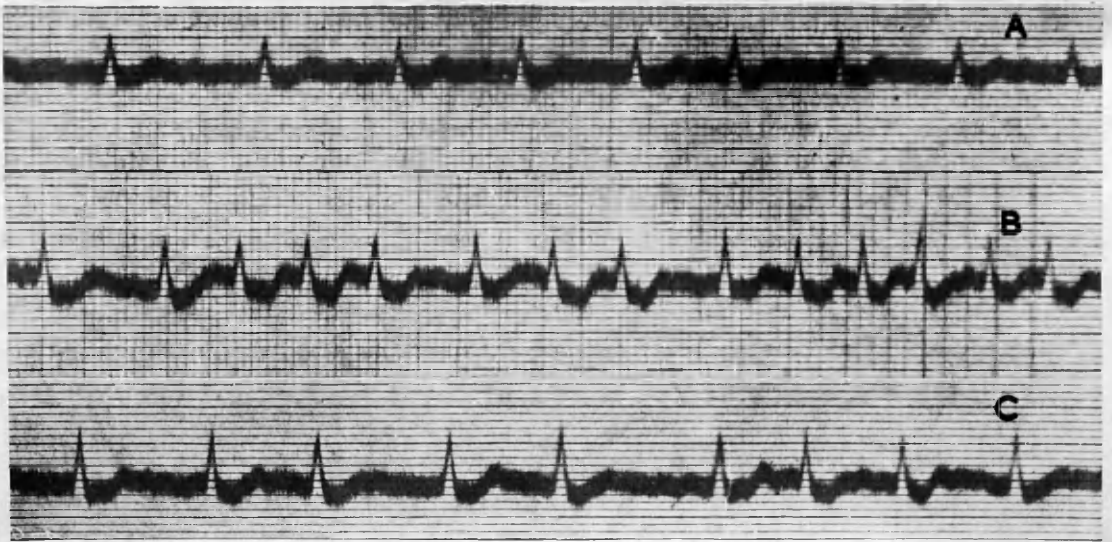


Fig. 63. Electrocardiograms (string galvanometer) from a case of auricular fibrillation (Case C 48).

- A - lead I, before exercise.
- B - lead I, immediately after standard step test.
- C - lead I, 15 seconds after B.

Series S.T.C. 10 climbs with 30 kg. load.

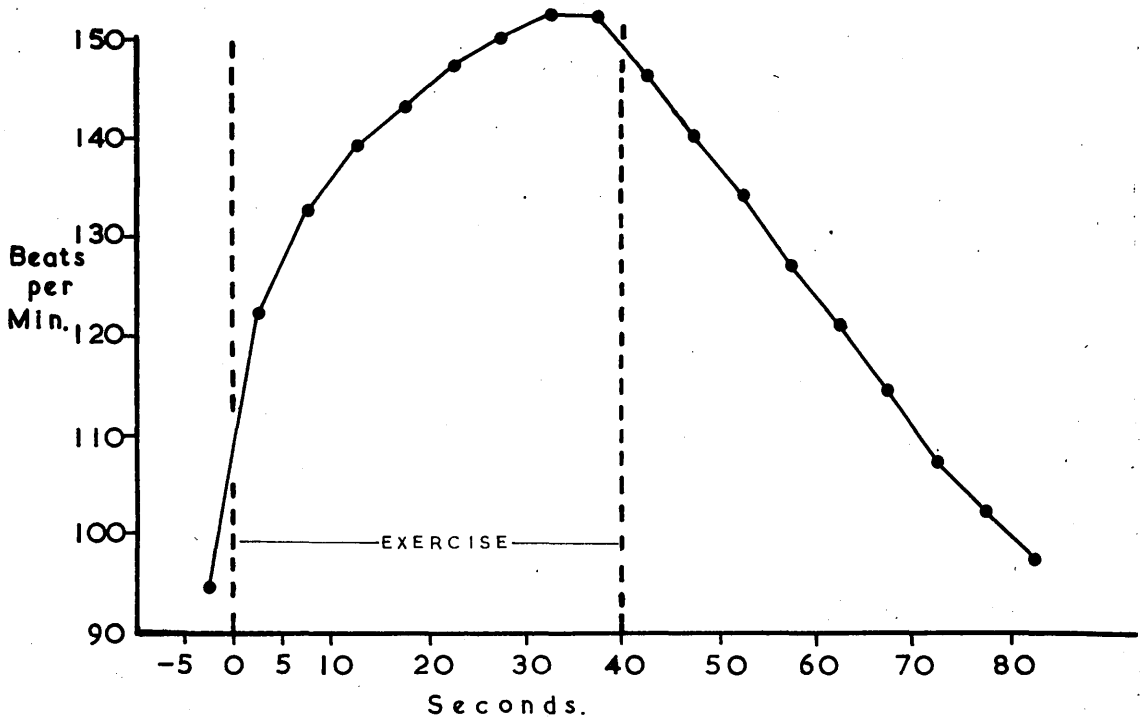
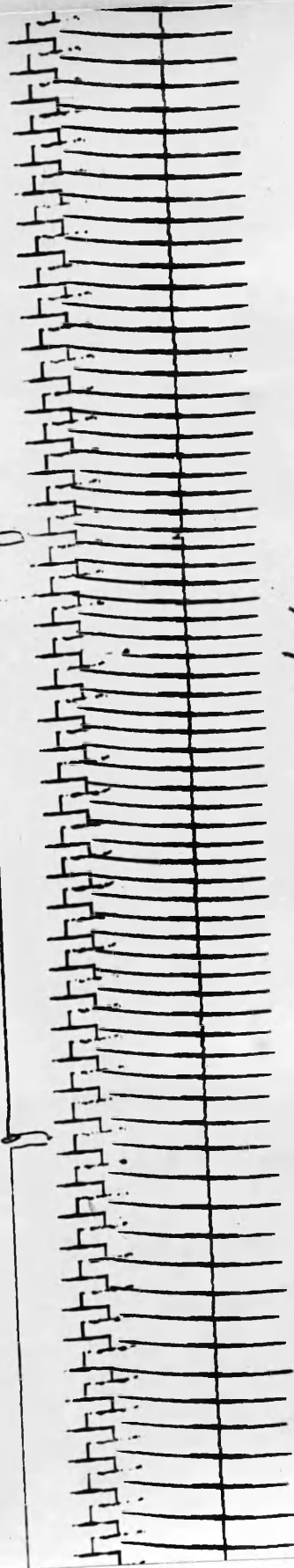


Fig. 64. Series S.T.C. (45 subjects).

Graph of mean heart rate in 5 second intervals before, during and after exercise.

The exercise consisted of ten ascents of the two steps at 96 steps per minute, carrying a load of 30 kg.

5 x 4. 096/min



BEFORE

EXERCISE

18/5/43

Fig. 65. Subject, D.M. Wright.
Kymograph tracing of heart beats before, during and
after exercise.

Upper line - signal.
Middle line - time in seconds.
Lower line - heart beats.

Between the dips in the signal line the subject
performed the standard two-step test.

Wright. Step Test before and after a short run on the treadmill.
Series A normal curve for comparison.

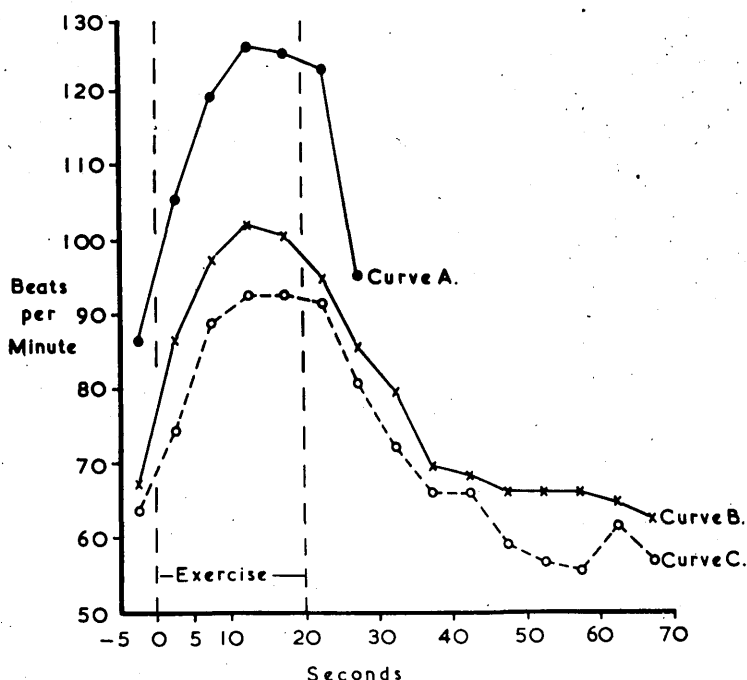


Fig. 66. Subject D.M. Wright.

Effect of a short run on the treadmill on the response of the heart rate to the standard step test.

Curve A - mean heart rate curve of 75 male medical students, for comparison.

Curve B - typical heart rate curve for Wright before a run on the treadmill.

Curve C - heart rate curve for Wright ten minutes after a half-mile run on the treadmill.

Wright. Standard step test before and after a run of 5.6 miles
on the treadmill.

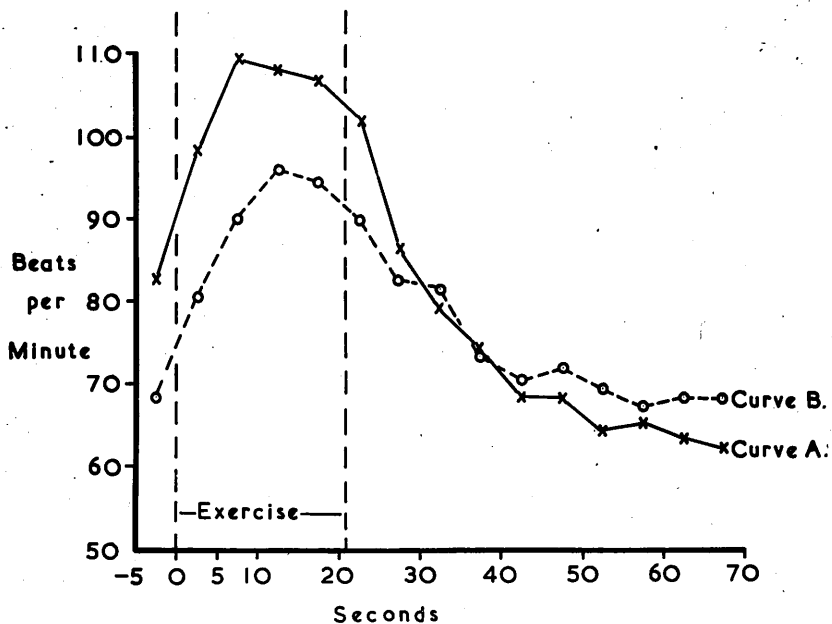


Fig. 67. Subject D.M. Wright.

Effect of a run of 5.9 miles on the response of the heart rate to the standard step test.

Curve A - heart rate before, during and after step-test performed ten minutes before the run.

Curve B - heart rate before, during and after step-test performed ten minutes after the run.

(Erratum - the legend above the figure should read 5.9 miles, not 5.6 miles)

Fig. 68. Subject, D.M. Wright.
Kymograph tracing of heart beats and respiration
during the start of the six mile run on a motor-
driven treadmill.

Upper line - signal.
Second line - time in seconds.
Third line - heart beats.
Fourth line - respiration.

The actual commencement of running is indicated by
the vertical ink line.

START MARKING.

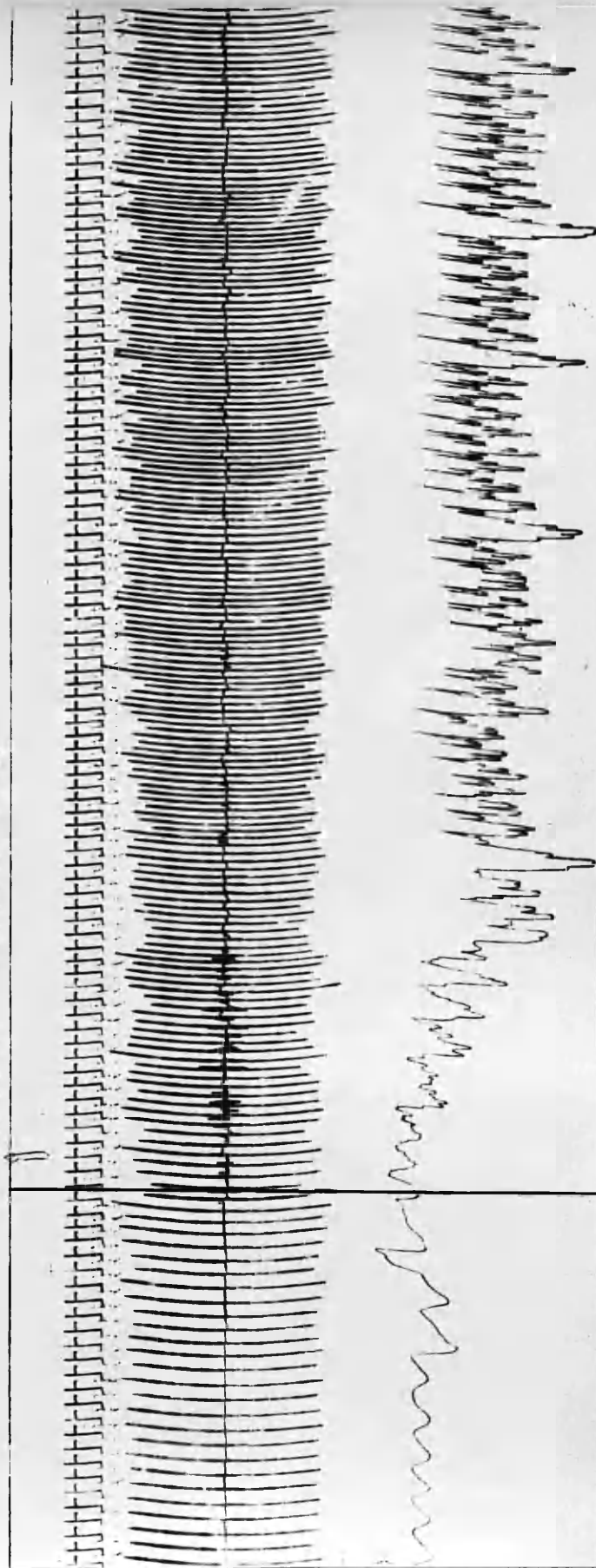


Fig. 68.

Wright. 5-6 mile run on treadmill.

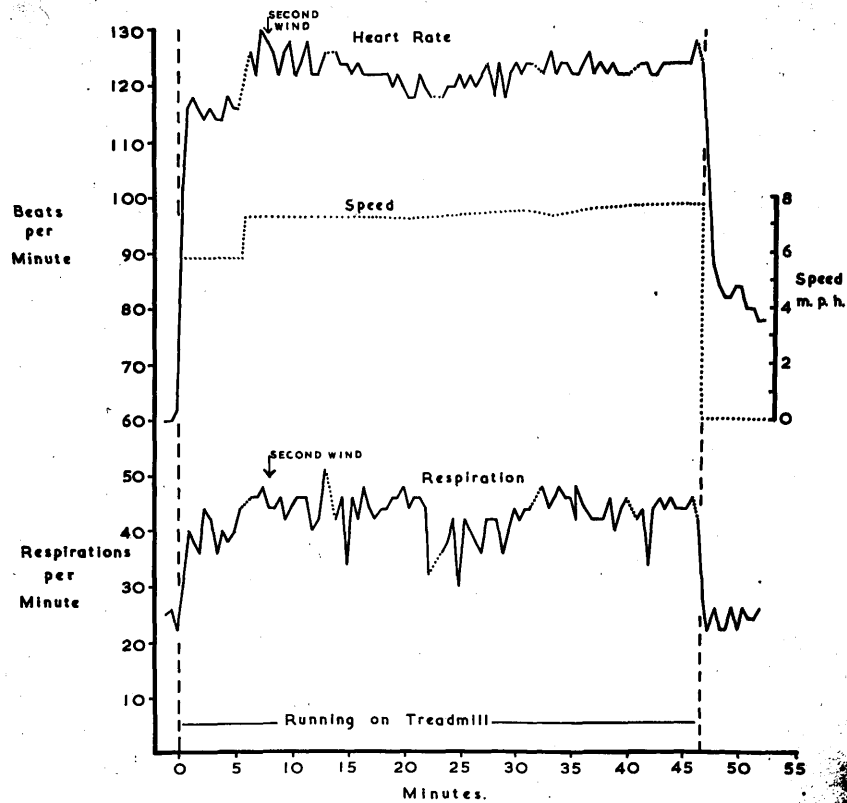


Fig. 69. Subject D.M. Wright.

Graphs of heart rate, respiration rate and speed of running throughout a 5.9 mile run on motor-driven treadmill.

(the dotted lines in the heart and respiration rate curves indicate breaks of 30 seconds during which the kymograph drum was changed).

Wright. Heart Rate during start of four runs.

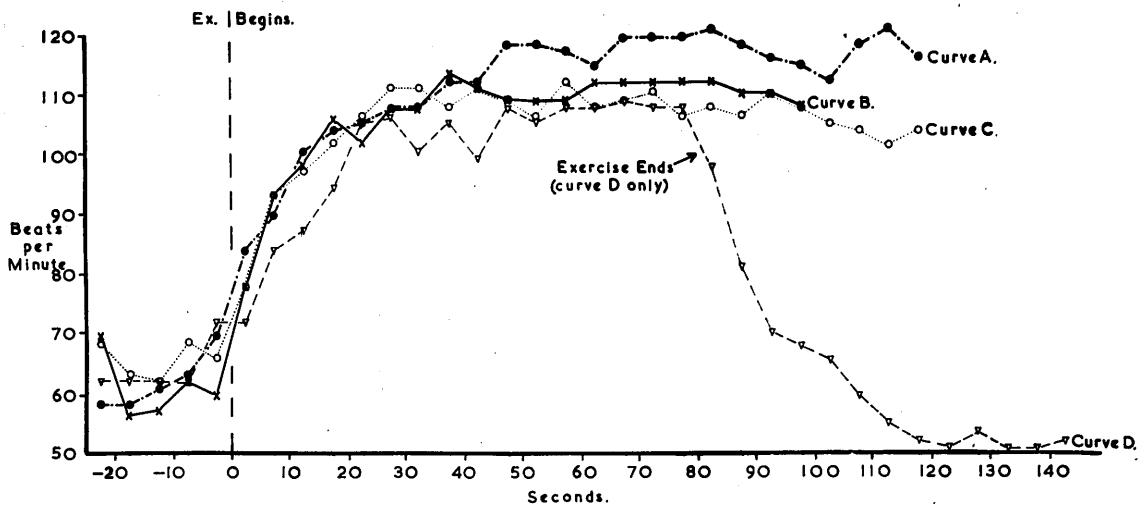


Fig. 70. Subject D.M. Wright.

Graphs of heart rate in 5 second intervals at the start of various runs on the treadmill.

Curve A - start of 5.9 mile run.

Curves B, C & D - three separate starts at half-hour intervals. Taken one year after Curve A.

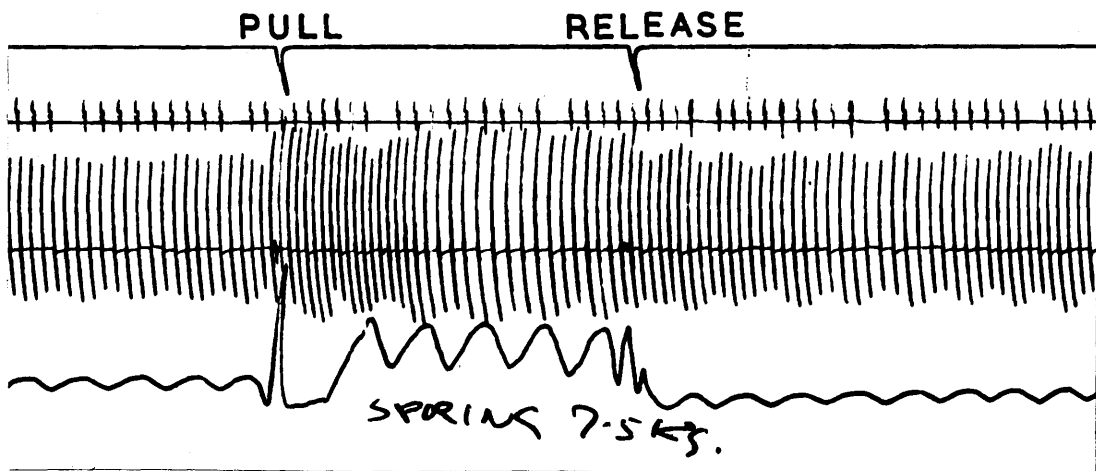


Fig. 71. Typical kymograph tracing of heart beats and respiration during a brief static effort.

Upper line -- signal.
 Second line - time in seconds.
 Third line -- heart beats.
 Fourth line - respiration.

At first signal mark subject pulled out two springs to a tension of 7.5 kg., and at second signal mark the springs were returned to zero.

Tracing shows 'initial' and 'release' accelerations of the heart rate.

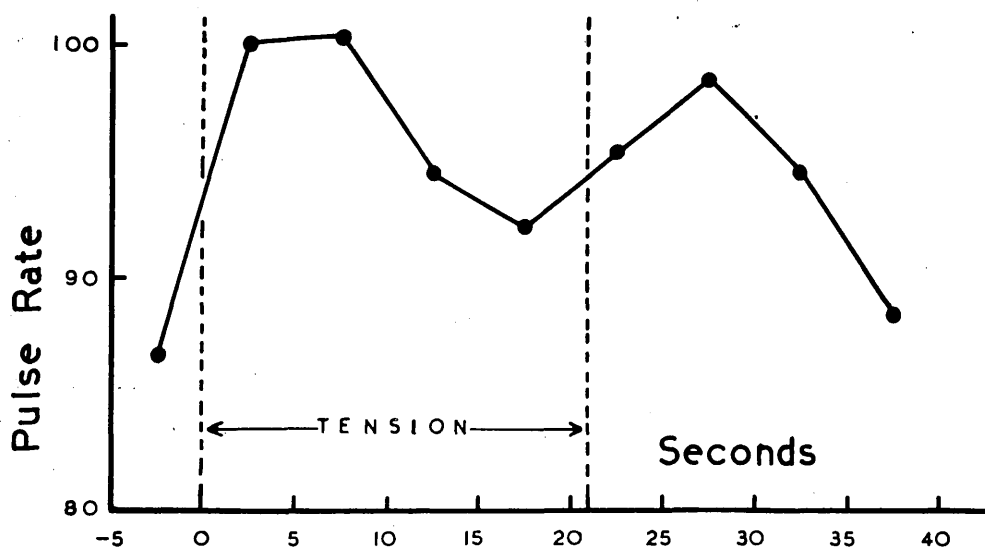


Fig. 72. Graph of mean heart rate in 5 second periods before, during and after a brief static effort.

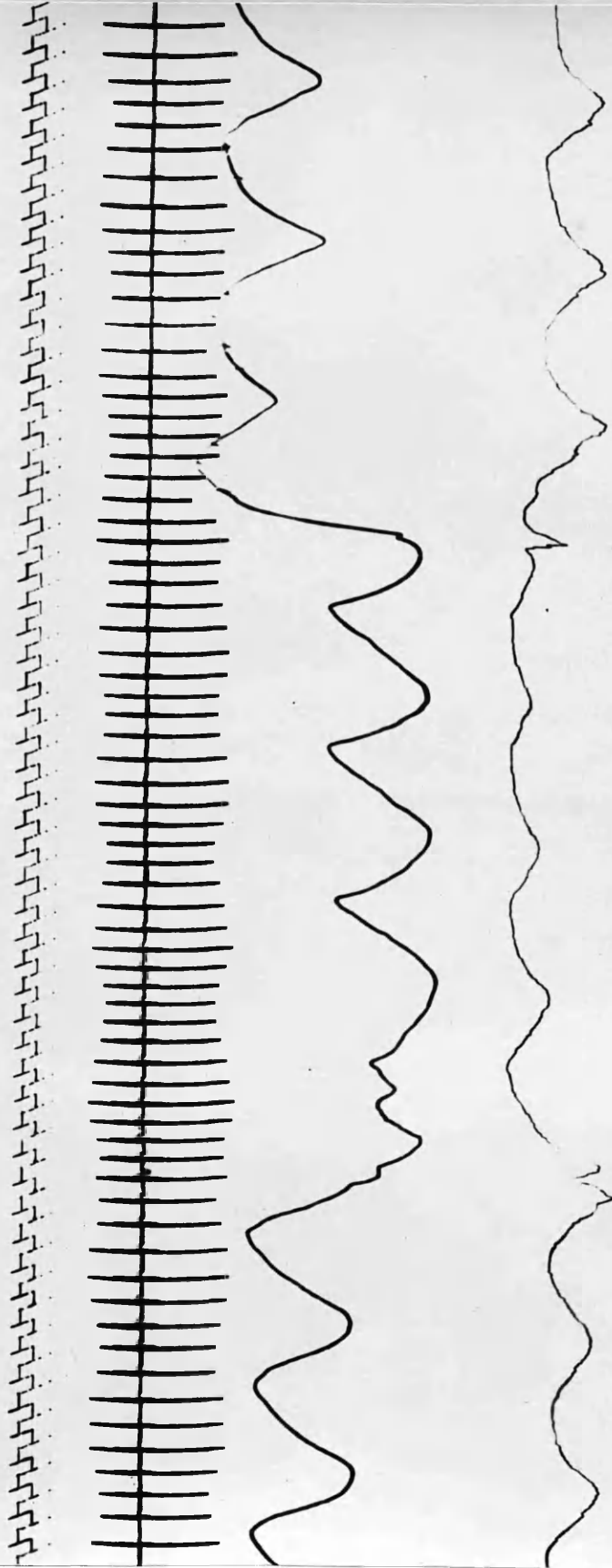
First series, eight subjects.

Fig. 73. Typical kymograph tracing of heart beats and respiration during static effort experiment. 7.5 kg. pull for 20 seconds between signal marks.

Upper line	-	signal.
Second line	-	time in seconds.
Third line	-	heart beats.
Fourth line	-	thoracic respiration (downstroke indicates inspiration).
Fifth line	-	abdominal respiration (downstroke indicates inspiration).

PULL

RELEASE



7.5-K ζ Pull-

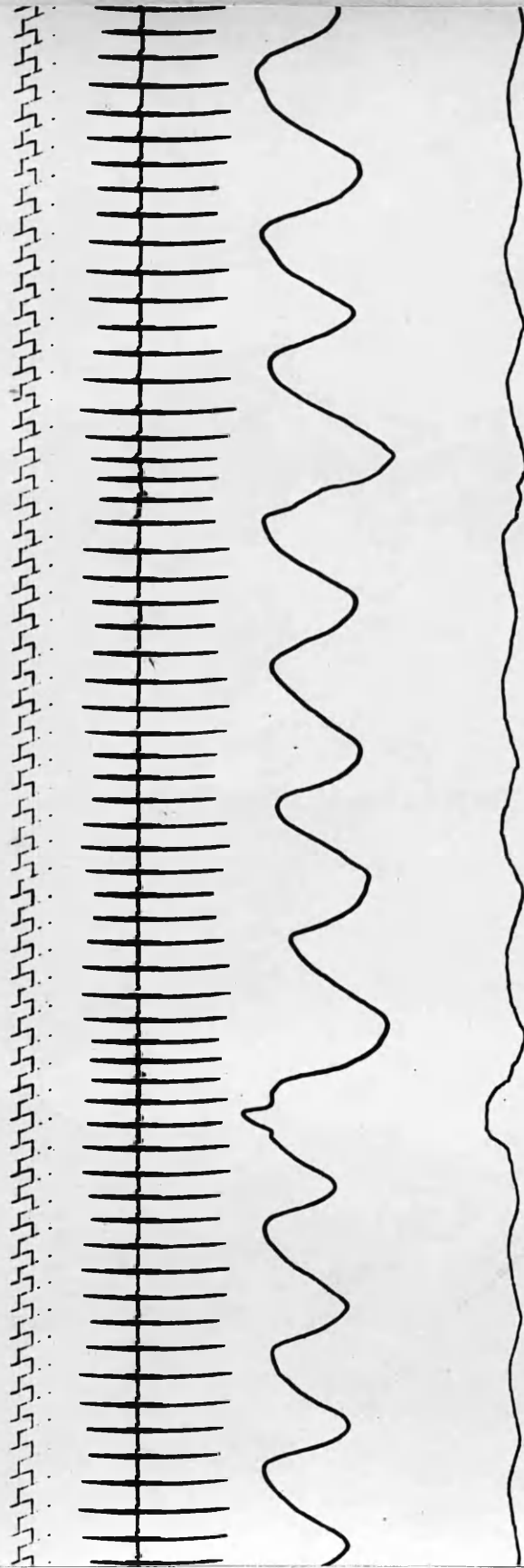
Fig. 73.

Fig. 74. Kymograph tracing of heart beats and respiration during 'sham static effort' experiment in same subject as in fig. 73. No tension on springs. Arm movements of pull and release as indicated on signal line.

Upper line	-	signal.
Second line	-	time in seconds.
Third line	-	heart beats.
Fourth line	-	thoracic respiration (downstroke indicates inspiration).
Fifth line	-	abdominal respiration (downstroke indicates inspiration).

PULL

RELEASE



SHAM SE

Fig. 74.

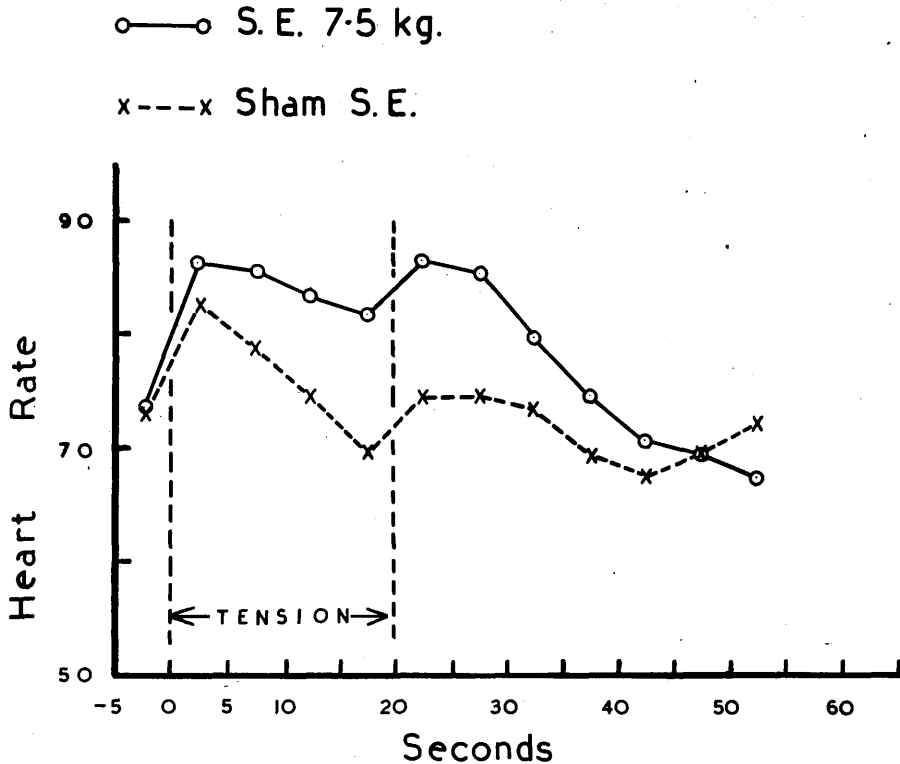


Fig. 75. Static effort; second series, fifteen subjects.

Upper curve (full line) - mean heart rates during pull of 7.5 kg.

Lower curve (broken line) - mean heart rates of same subjects during performance of the same movements, but without tension on the springs (sham static effort).

S.E. minus Sham S.E.

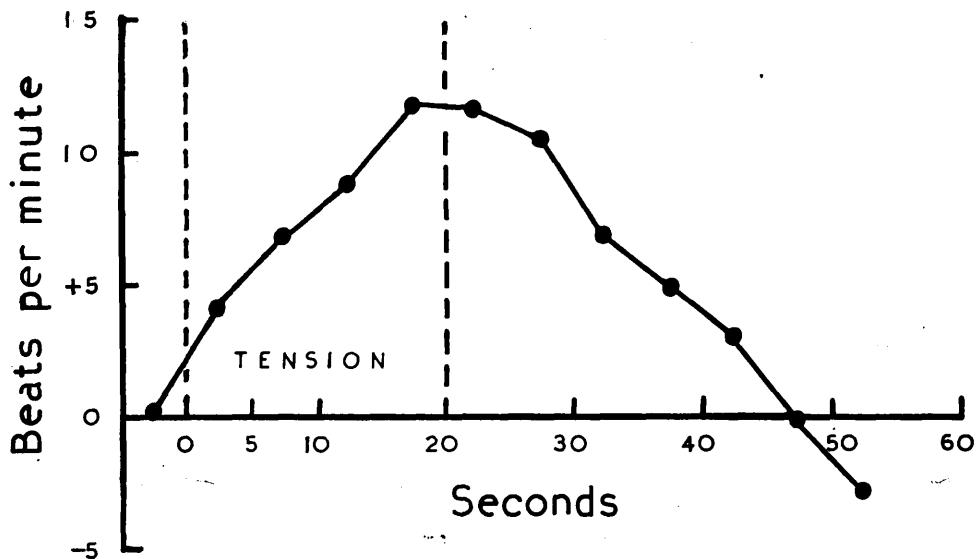


Fig. 76. Static effort, second series. Response of heart rate to static component alone.

Graph obtained by subtracting the heart rates in each 5 second period of the lower graph from those of the upper graph in Fig. 75.

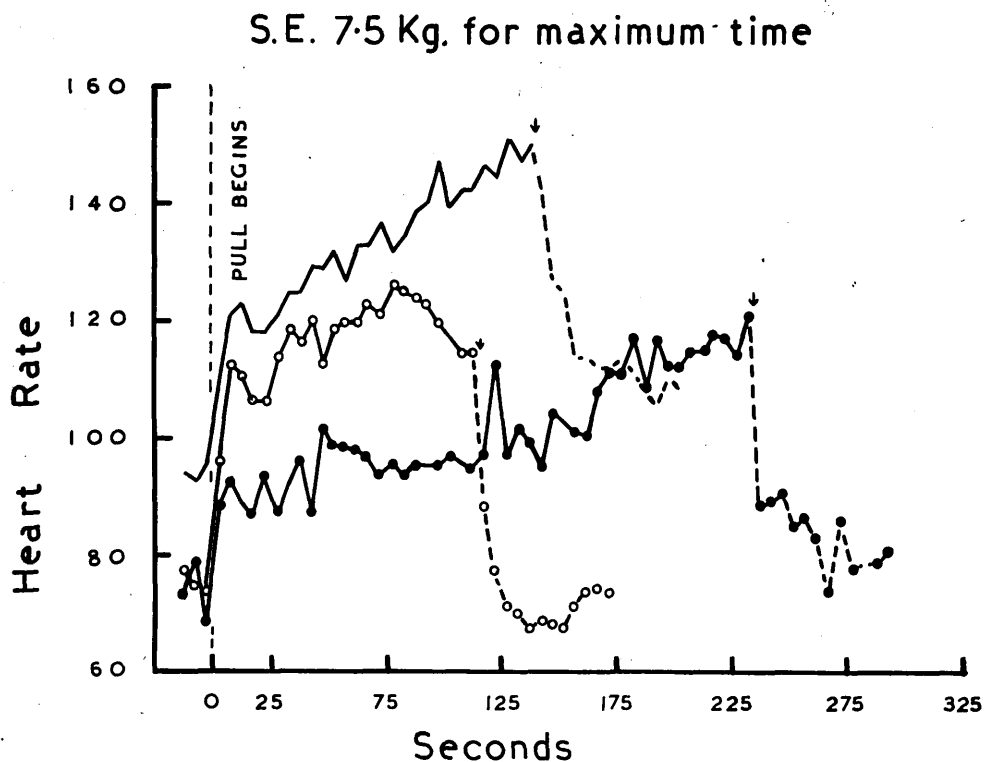


Fig. 77. Graphs of heart rates in 5 second intervals for three subjects performing a static effort of 7.5 kg. until they could no longer hold the arms steady.

Small arrows indicate the end of effort for each curve.

TABLE 1.

SUMMARY OF RESULTS IN SERIES A (75 men) AND SERIES B (25 women)
Standard Tolerance Test, 5 ascents of 2 steps. Rate 96 steps per minute. No load.

	M e a n		R a n g e		Standard Deviation		Coefficient of variation (percentage)	
	Series A (men)	Series B (women)	Series A (men)	Series B (women)	Series A (men)	Series B (women)	Series A (men)	Series B (women)
Early Resting Rate, beats/minute.	81.5	84.4	58-128	62-120	13.9	14.9	17.1	17.7
Initial Rate, beats per minute	85.7	90.4	59-121	67.2-119.4	13.8	14.6	16.1	16.1
Maximum Rate, beats per minute	130.2	132.6	109-168	103.2-159.6	12.9	13.7	9.9	10.3
Time to reach Maximum Rate (seconds)	15.0	17.1	7.5-22.5	7.5-27.5	-	-	-	-
Acceleration of Heart Rate (beats/min/sec)	3.0	2.61	1.4-5.3	1.03-5.45	0.9	1.0	28.7	36.8
Extra Beats	17.0	16.4	8.6-27.2	8.5-23.3	4.9	3.4	28.6	20.8
Percentage increase on initial rate	53.9	48.4	19.8-100	20.0-76.0	18.5	14.5	34.3	29.9

TABLE 1 (Continued).

SUMMARY OF RESULTS IN SERIES A (75 men) AND SERIES B (25 women)
Standard Tolerance Test, 5 ascents of 2 steps. Rate 96 steps per minute. No load.

	M E A N		R a n g e		Standard Deviation		Coefficient of variation (percentage)	
	Series A (men)	Series B (women)	Series A (men)	Series B (women)	Series A (men)	Series B (women)	Series A (men)	Series B (women)
Actual increase in beats per minute	44.4	42.3	25.2-69.6	21.0-60.6	10.1	9.1	22.8	21.4
Post-exercise rate, beats per minute	91.7	98.2	60-134	72.0-130.0	14.8	15.5	16.2	15.8
Time to return to normal (seconds)	21.4	25.4	5.0-71.0	7.5-44.5	16.5	9.4	77.1	36.8
Rate 5 min. after exercise, beats/minute	80.3	82.2	50-120	60-112	11.1	14.6	13.8	17.8
Duration of Exercise (seconds)	24.05	25.2	19-30	21-30	-	-	-	-

TABLE 2.

Summary of Literature on Resting Pulse Rate in Sitting Posture in Subjects of Comparable Age Group.

Pulse Rate (men)	Pulse Rate (women)	Authority	Remarks
71/min.	-	Volkman (1850)	Used his own observations and those of Guy & Nitzsch. Average age of subjects 20-24 years.
80/min. with back leaning 87/min. sitting upright	-	Langowoy (1900)	Small series of 20 experiments.
78/min.	82/min.	Guy & Steffen quoted in Vierordt (1906)	Men and women were in age group 14-21 years.
-	(athletic) 76/min. (non-athletic) 78/min.	Hartwell & Tweedy (1913)	Series of 16 athletic and 38 non-athletic women. Average age 21.5 years.
70.1/min.	-	Schneider (1920)	Quoted as general average.
-	80/min.	Burlage (1921)	Series of 1700 girls of average age 18 years.
74.7/min. (range 52-106)	79.5/min. (range 53-116)	Hambly et al. (1922)	406 observations on 94 men and 103 observations on women.
-	84.46/min.	Cripps (1924)	Series of 26 women of average age 19 years before physical training.
78.4 (winter) 72.8 (summer)	-	Hill, Magee & Major (1937)	Series of 29 students, athletic, age group 21-26.
70-72/min.	75-81/min.	McCurdy & Larson (1939)	General average.

TABLE 3.

Series A.

Comparison of Exercise Tolerance Indices in Subjects with and without marked Sinus Arrhythmia.

I n d e x	Mean value in 10 subjects showing marked Sinus Arrhythmia	Mean value in 10 subjects showing no Sinus Arrhythmia
Early Resting Rate (beats/min.)	89.0	79.8
Initial Rate (beats/min.)	95.5	83.7
Maximum Rate (beats/min.)	135.0	123.5
Time to reach the Max. Rate (seconds)	13.0	15.0
Acceleration of Heart Rate (beats/min/sec)	3.42	2.54
Extra Beats	14.8	14.7
Actual Increase in Heart Rate (beats/min)	39.5	39.8
Percentage Increase over Initial Rate	43.5	48.8
Post-Exercise Rate (beats/min.)	97.8	87.0
Return to Normal (seconds)	18.4	17.3
Rate 5 mins. after Exercise (beats/min)	84.7	79.0
Age in Years	20.3	20.4

TABLE 4.

Variation of Indices with Load carried. Series W.
 (means of observations on 17 subjects). Number of climbs
 (5) constant. Rate of climbing constant (96 steps/minute).

I n d e x	NO LOAD	LOAD 19.5 kg.	LOAD 32.2 kg.	LOAD 40.3 kg.
Initial Rate (beats/min.)	86.7	87.4	88.9	90.1
Maximum Rate (beats/min.)	124.9	130.9	134.3	137.4
Time to reach Max. Rate (seconds)	14.6	18.1	18.4	19.3
Acceleration of the Heart Rate (beats/min/sec)	2.7	2.5	2.7	2.6
Extra Beats	19.2	25.3	28.1	33.4
Percentage Increase on Initial Rate	45.3	51.3	52.5	55.1
Actual Increase in beats/min.	38.2	43.5	45.3	47.2
Post-exercise rate (beats/min.)	97.9	104.9	111.1	116.4
Time to return to normal (seconds)	32.0	36.8	50.6	56.0
Duration of Exercise (seconds)	20.4	21.4	21.9	22.4
Mean Total work (kg.m.)	163	212	244	266
Mean Rate of Working (kg.m. per min.)	478	595	669	712

TABLE 5.

Summary of Results of Standard Tolerance Test (5 climbs at 96 steps/min. No load) in the Load and Duration Series. Total of 30 male subjects. For comparison with results in Series A (Table 1).

I n d e x	Mean	Range	Corrected Standard Deviation	Coefficient of Variation (percentage)
Initial Rate (beats/min.)	83.8	58-112	14.5	17.3
Maximum Rate (beats/min.)	123.3	102-160	14.9	12.1
Time to reach Maximum Rate (secs.)	15.0	7.5-22.5	-	-
Acceleration of heart rate (beats/min/sec)	2.7	1.29-4.03	0.57	21.1
Extra Beats	19.3	7.4-31.4	7.2	37.4
Percentage increase on initial rate	49.2	27.9-94.6	15.7	31.9
Actual increase in beats/min.	39.5	24.8-48.8	-	-
Post-exercise rate (beats/min.)	94.8	80-136	16.7	17.6
Time to return to normal (seconds)	30.9	6.5-137	25.2	81.6

TABLE 6.

Variation of Indices with Duration of Exercise. Series D.
(means of observations in 13 subjects). No Load. Rate of
climbing constant (96 Steps/min.)

I n d e x	5 CLIMBS mean total work 167.5 kg.m.	10 CLIMBS mean total work 335 kg.m.	20 CLIMBS mean total work 670 kg.m.
Initial Rate (beats/min.)	80.0	79.1	76.2
Maximum Rate (beats/min.)	121.3	127.7	133.9
Time to reach maximum rate (seconds)	15.6	29.4	61.7
Acceleration of the heart rate (beats/min/sec)	2.7	1.7	0.9
Extra Beats	19.6	38.9	77.3
Percentage increase on Initial Rate	54.3	65.3	79.8
Actual Increase in (beats/min.)	41.3	48.6	57.8
Post-exercise rate (beats/min.)	90.8	97.4	102.6
Time to return to normal (seconds)	29.4	36.9	47.1
Duration of Exercise (seconds)	20.9	40.5	78.6

TABLE 7.

Variation of Indices with Speed and Duration of Exercise. Series S.
(means of observations on 10 subjects). No load.

I n d e x	5 climbs at 96/min sitting to sitting	5 climbs at 48/min standing to standing	10 climbs at 48/min standing to sitting	5 climbs at 96/min standing to standing	10 climbs at 96/min. standing to standing
Initial Rate (beats/min.)	85.7	93.8	95.4	95.6	94.6
Maximum Rate (beats/min.)	118.1	115.6	117.0	122.3	130.0
Time to reach Maximum Rate (secs)	13.5	25.0	55.0	16.5	33.0
Acceleration of the heart rate (beats/min/sec)	2.6	0.86	0.45	1.67	1.04
Extra beats	12.6	7.7	6.0	9.9	11.1
Percentage increase on initial rate	39.9	24.2	23.8	29.0	39.4
Actual increase in (beats/min.)	32.4	21.7	21.6	26.7	35.4
Post-exercise rate (beats/min.)	94.4	99.2	100.6	103.4	111.2

TABLE 7 (continued).

Variation of Indices with Speed and Duration of Exercise. Series S.
(means of observations on 10 subjects). No load.

I n d e x	5 climbs at 96/min sitting to sitting	5 climbs at 48/min standing to standing	10 climbs at 48/min standing to sitting	5 climbs at 96/min standing to standing	10 climbs at 96/min standing to standing
Time to return to normal (seconds)	27.0	22.8	30.6	24.9	37.4
Duration of exercise (seconds)	19.8	36.2	72.6	18.5	37.5
Mean Total Work performed (kg.m.)	182.3	182.3	364.6	182.3	364.6
Mean Rate of Working (kg.m. per minute)	556	302	301	592	584

TABLE 8.

Series C. Details of the 12 patients in group V.D.H.

No.	Sex	Age	Lesion	Remarks	Tolerance Group
C7	M	13	Congenital Pulmonary stenosis and septal defect	No symptoms, no cardiac enlargement, E.C.G. Slight R.V. preponderance.	Excellent
C13	F	15	Congenital Pulmonary stenosis	Occasional palpitation. Tachycardia. No limitations on exercise. E.C.G. Biphasic R waves. Tl inverted.	Excellent
C1	M	18	Mitral Incompetence	Followed rheumatic pericarditis. In employment. Symptomless except on severe exertion.	Very good
C11	F	15	Mitral stenosis and incompetence.	Rheumatic Fever. Persistent tachycardia. In employment.	Very good
C25	M	45	Aortic Incompetence.	In employment. Symptoms only on severe exertion.	Very good
C26	M	23	Mitral stenosis.	Not in employment. Dyspnoea only on moderately severe exercise.	Good
C41	M	20	Mitral incompetence.	Student. Strained heart hill climbing. Symptoms only when walks fast up hill.	Good

TABLE 8 (continued).

Series C. Details of the 12 patients in group V.D.H.

No.	Sex.	Age	Lesion	Remarks	Tolerance Group
C38	F	20	Mitral incompetence	Not in employment. Dyspnoea and substernal pain when hurries but not on ordinary walking.	Fair
C19	F	40	Mitral and Aortic Lesions.	Dyspnoea and palpitation when walks quickly and climbs stairs. Not on normal walking.	Fair
C39	M	36	Mitral and Aortic Lesions.	Dyspnoea and substernal pain when hurries and on stairs. Not on level walking.	Fair
C27	F	38	Mitral incompetence	Breathless on stairs. Not on level.	Fair
C31	M	17	Mitral stenosis.	Not in employment. Dyspnoea and precordial pain on rapid walking. Not on ordinary walking on level.	Fair

TABLE 9.

Series C. Group V.D.H. Indices grouped by Tolerance Estimate.

I n d e x	Tolerance Estimate			
	Excellent (2 cases) Means	Very Good (3 cases) Means	Good (2 cases) Means	Fair (5 cases) Means
Early resting rate (beats per minute)	98.0	101.3	95.0	90.4
Initial Rate (beats per minute)	98.1	99.6	94.5	91.4
Maximum Rate (beats per minute)	126.0	129.6	128.4	132.5
Time to reach Max. Rate (seconds)	12.5	17.5	25.0	19.5
Acceleration of Heart Rate (beats/min/sec)	2.16	1.70	1.52	2.21
Extra Beats	9.5	10.9	14.3	16.3
Percentage Increase on Initial Rate	28.5	31.6	36.1	46.8
Actual Increase in beats per minute	27.9	30.0	33.9	41.1
Post-exercise rate (beats per minute)	104.0	109.3	112.0	117.2
Time to return to normal (seconds)	21.0	28.2	42.0	51.5
Duration of Exercise (seconds)	22.5	20.3	20.0	21.7

TABLE 10.Series C and B.C. Details of Auricular Fibrillation patients.

No.	Sex.	Age	Remarks	Tolerance Group
C9	M	32	Simple A.F., on digitalis. Can take walks and play golf. Not in employment.	Good
C40 & 45	M	47	Mitral stenosis, on digitalis. Breathless on long stairs.	Fairly Good
C48	F	36	Simple A.F., on digitalis. Breathless on long gradients.	Fairly Good
C2	M	26	Mitral stenosis, not on digitalis. Breathless on stairs.	Fair
C21	F	37	Mitral stenosis, on digitalis. Breathless on stairs.	Fair
C49	M	37	Simple A.F., on digitalis. Breathless on stairs.	Fair
C5	M	29	Patent Ductus Arteriosus and A.F., not on digitalis. Easily tired.	Poor
C10 & 22	M	22	Mitral stenosis, on digitalis. Breathless on slight exertion.	Poor
C18	F	43	Coarse fibrillation, on digitalis. Cyanotic.	Poor
C20	F	44	Coarse fibrillation, not on digitalis. Easily tired.	Poor
C30	F	33	Mitral stenosis, not on digitalis. Cyanotic on slight exercise.	Poor
C46	M	48	Simple A.F., on digitalis. Breathless on slight exertion.	Poor
C47	M	40	Mitral incompetence, on digitalis. Breathless on level.	Poor

TABLE 10 (continued).

Series C and B.C. Details of Auricular Fibrillation patients.

No.	Sex	Age	Remarks	Tolerance Group
B.C. 16-24	M	34	Simple A.F. Orthopnoeic on admission. Serial tests made during treatment.	In hospital
B.C. 26	M	32	Same patient as C5. Patent Ductus Arteriosus. On digitalis.	In hospital
B.C. 27	M	45	Mitral incompetence, rheumatic carditis. Not on digitalis.	In hospital

Table 11. Series C. Auricular Fibrillation Patients. Means of heart rate indices grouped by Tolerance Estimate.

	T O L E R A N C E E S T I M A T E				
	GOOD	FAIRLY GOOD	FAIR	POOR	
	On digitalis (1 case)	On digitalis (2 cases)	On Digitalis (2 cases)	Not on digitalis (1 case)	Not on digitalis (3 cases)
Early Resting Rate (beats/min.)	68	109	94	132	103
Initial Rate (beats per minute)	66.6	115.8	95.7	135.6	99.5
Maximum Rate (beats per minute)	129.6	184.8	151.2	235.2	155.4
Time to reach Maximum Rate (secs)	32.5	25.0	20.0	17.5	17.5
Acceleration of Heart Rate (beats/min/sec)	2.33	2.21	2.80	5.97	3.13
Extra Beats	18.7	21.6	18.2	24.2	17.8
Percentage Increase on Initial Rate	94.5	59.5	59.0	78.0	58.9
Actual Increase in beats per minute	63.0	69.0	55.5	99.6	56.0
Post-Exercise Rate (beats per minute)	104.0	140.0	132.0	176.0	134.5
Duration of Exercise (seconds)	22.0	22.5	22.5	21.0	23.0
					22.2

TABLE 12.

Summary of Results of Special Tolerance Test (10 climbs at 96 steps/min. load 30 kg.) in Senior Training Corps Series. Total of 45 male subjects.

Index	Mean	Range	Standard Deviation	Coefficient of Variation (percentage)
Initial Rate (beats/min.)	93.3	58-140	20.2	21.7
Maximum Rate (beats/min.)	155.0	124.8-192.0	14.9	9.6
Time to reach Maximum Rate (secs.)	33.5	12.5-57.5	-	-
Acceleration of heart rate (beats/min/sec)	1.90	0.76-3.84	0.58	30.5
Percentage Increase on Initial Rate	70.7	21.6-157	33.7	47.7
Actual Increase in Beats/min.	61.1	31.2-92.4	14.9	24.3
Post-Exercise Rate (beats/min.)	124.4	90-170	18.6	14.9
Time to return to normal (seconds)	81.0	45-195	-	-
Duration of Exercise (secs.)	39.8	35-45	-	-
Total work (kg.m)	490	-	-	-
Rate of Working (kg.m./min.)	742	-	-	-

TABLE 13.

Instructor's Estimate of Physical Condition of
Series S.T.C. subjects, and some heart rate indices from
the exercise test.

I N D E X	PHYSICAL FITNESS ESTIMATE		
	Above Average (Mean of 14 subjects)	Average (Mean of 19 subjects)	Below Average (Mean of 8 subjects)
Initial Rate (beats/min.)	87.4	87.7	118.0
Range of Initial Rate	58.2-123.0	56.4-110.4	96.6-144.0
Maximum Rate (beats/min.)	148.7	149.3	177.2
Range of Maximum Rate	130.8-170.4	124.8-168.0	168.0-192.0
Actual Increase (beats/min.)	61.3	61.6	59.2
Percentage Increase	70.2	70.3	50.2